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The Beattie-Smith Lectures.[1]

(THE UNIVERSITY OF MELBOURNE.)

THE CONDITIONS OF CIVILIZED LIVING AND THE PROBLEMS OF MENTAL HEALTH.

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LECTURE II: THE TRAINING AND INTERRELATIONS OF PSYCHOLOGISTS, PSYCHIATRISTS AND GENERAL PRACTITIONERS.

THE first lecture tried to show by various examples that the social relations and adjustments of the human being, though exceedingly complex, can be grouped under four main headings: social institutions, roles, social relations, and personality or internalized culture. These four categories form a scale of decreasing rigidity, or increasing liability to fluctuation, in the constant shifting of dynamic equilibria both within an individual and between individuals.

The central aim of that lecture was to consider the phenomena of mental health from a new point of view.

We saw in the genesis of prejudice—for instance, prejudice and "scapegoatitis" among front-line troops against groups of people at home—that the functional role participation of people is disturbed so that they see themselves in relation to others differently, and that the mechanism of projection plays a part in mental epidemiology.

Case Discussions.

Having considered early psychological disorders on, as it were, a large scale, let us now look at one or two individual cases, chosen because they so clearly illustrate the influence of social forces on individual health.

The first case is a very simple prototype of psychosomatic disorder. From it we could go on to severe disorders such as asthma and peptic ulcers.

A girl, aged eight years, was refusing to eat her evening meal and beginning to leave her school lunches untouched. She also refused to go to sleep and often lay awake until far into the night. She lost weight and was pale and listless. The doctor prescribed tonics and a light diet, which made no difference. A psychologist suggested that the mother and father should cease to attempt coercion either in the food situation or when the girl was in bed. When this advice was followed, a "dramatic" cure resulted.

But it is not the dramatic aspect I want to emphasize. In this Scottish household, the parents saw the child through their role as authoritarians. The first duty of a child is to obey its parents. The headmaster of a famous Scottish high school once attended a lecture I gave on "progressive schools", and in the course of an argument with me defined education thus: "Education is the inculcation of a respect for authority." The child, however, saw herself in the role of a child neglected in favour of her younger sister. She wanted to draw attention to herself. She got attention, but with it stress and hostility. The parents, faced with a situation they did not understand, reacted in their familiar role—parent commands, child obeys. The doctor acted in the only role he knew: to stimulate the taste, tone up the system, get the food inside somehow, and let the digestive processes do the rest.

The next case concerns a high-ranking naval officer who, on arriving at a base in Britain after an absence of some four years, developed an acute and protracted illness before being able to go on leave. At that base hospital were assembled many of Britain's foremost specialists and the officer had the best possible treatment. He was looked after by a magnificent nursing staff, who gave him every treatment that scientific ingenuity had devised. Several other specialists were called into consultation.

[1] Delivered at Melbourne on June 19 and 26, 1950.

At the height of the crisis, his wife was summoned from Cornwall. When she arrived after an intolerably long wartime train journey, the crisis had passed and he was again conscious, but very weak.

Before being shown into his room, the wife was "briefed" by the matron and the physician in charge of the case. The patient's recovery, they said, was being slowed by his unwillingness to eat. The wife was asked to exert her influence and get him to eat more. On her first visit, therefore, she tried to carry out her instructions. The result was that he suffered a relapse.

You may say that either the husband or the wife or both needed psychiatric treatment. This was not the view of a psychiatrist, a friend of the officer, who visited him later. He saw it as a failure in the training of the matron and the doctors. None had learnt to think in terms of social psychology, though their handling of the "case"—and further their *rapport* with the patient—left nothing to be desired. Their success in pulling him through proved that.

Let us consider the social psychology of this case.

The patient was not a regular naval officer, but in civil life a professional man. For four years he had been pushed about by the navy over the seven seas. He was growing tired of being pushed about. His wife belonged to an old Bloomsbury family. She was intelligent and charming, a delightful mother to their three children. But she had thoroughly identified herself with Bloomsbury values. That is, emotions were something to be discussed and analysed. In the civilized human being, the cerebral cortex, they felt, was dominant, and life was to be ordered on rational, scientific principles. Now in the present highly stressful situation she was told by two medical authorities that what the patient needed was physical building up, through the ingestion of greater quantities of food; that the nurses were not able to impress this on him; but that a wife would surely be able to succeed. What could be more eminently reasonable and "common-sensical"? But after weeks of delirium the patient had emerged from the stage of being merely a sick body. To his mind the wife now appeared in the role of one who had identified herself with authority. In any case, after four years of disconnexion from civilian society, his perception of family roles had also weakened. His fantasies of a young, beautiful, all-loving wife could not, in his weakened state, stand the shock of reality: a real wife—not a ministering angel—tense with anxiety and travel, and a mere handmaiden of authority, another nurse.

Three histories converge to this crucial point in a man's life. His own, which included his four years of gradually attenuating social connectedness and increasing resentment against the war and military authority; his wife's, which included her identification with a particular European rationalistic subculture and her also war-attenuated social role of wife; and the history of the training of the physician.

His training had given him superb skills in the diagnosis and treatment of physical disease; yet his lack of insight into the forces of social psychology had nearly brought his skills to nought. Let us inquire whether this lack was a result of incomplete training, or was an anopsia induced by social forces.

Some Pointers from the History of Medicine.

Once upon a time, the doctor was a priest—as he still is among primitive peoples. And the function of a priest is to relate man to a greater cosmos. Not parts of a man, but the whole man, or at any rate the whole soul.

Stefan Zweig (1931) has written a book about three very different "healers"—Freud, Mesmer and Mary Baker Eddy. His introduction, though somewhat imaginative, puts the matter so well that I shall follow his main lines of exposition.

Religion, Zweig says, comes about through the fact of suffering, in this way: health just is, and needs no explaining; but illness falls upon a man suddenly, bringing with it contradictory feelings of hope and despair, rebellion and resignation, guilt and innocence, fear and faith. The senselessness of illness cries out for explanation. And—as we should now put it—the mechanism of projection is used: someone must be angry with me, hate me; someone with powers transcending those of mortals. The gods send illness, and only they can take it away. They can be reached only through a priest.

In the beginning, then, the fight of the healer was not against the disease, but was an effort in applied theology. Every device of cult, ritual and magic was used—hence the secrecy with which the healer's art was surrounded. But no theological device could be successful unless the patient and his family, in conjunction with the priest, devoted themselves to prayer and meditation. In other words, crucial to the conception of healing was the establishment of a unity of feeling, of active participation in a small social group, for the purpose of relating this group to the governing forces of the universe.

But though man may be an incurable mystic, he is also an incurably rational being. With the development of special knowledge and experience of sick people, the doctor began to emerge as a specialist. At first, he was with priest and patient, later he came between them. Against transcendentalisms he set the use of worldly things, of herbs, powders and liquors; against blind belief he set open-eyed reason. The unity of the therapeutic group was broken. The doctor attended to earthly things, the priest to heavenly.

The next development was to split up diseases into component parts, into a host of separately observable phenomena. Disease became not something that happens to a person, only something that happens to organs. Zweig quotes the great pathologist Virchow as saying at a medical congress in Rome: "There are no general diseases, only diseases of organs and cells." The task of the doctor now became diagnostic and classificatory. A given confluence of signs and symptoms was categorized as a diseased type, and this determined treatment. A calling became a profession; the mystery of healing became a craft, a science. Spontaneity, integration with the total feeling quality and aspirations of a small group, were replaced by what was learnt and calculable.

During the nineteenth century this split between patient and healer became intensified. Between the doctor and the patient there had already been inserted the cold, calculating attitude of the scientist. Now the instruments, the apparatus for diagnosis, also came between them; and, where previously the doctor had calculated and made up his own prescriptions in doses to give to the individual—based maybe on astrological considerations, but mainly because they were meant for the unique individual—now he got his prescriptions from books and his pills from factories.

This industrial rationalization, of course, is not to be blamed altogether on the doctor. He is himself a part of his society, from which, like everyone else, he absorbs, half unconsciously, the prevailing values and *mores*. These were the times when the triumphs of the natural sciences stimulated the belief that only those things are real which can be seen and dissected. In the industrial field the drift to the cities and the destruction of craftsmanship assumed catastrophic proportions and mass production began. Towards the end of the nineteenth century the moving belt brought new triumphs out of the factories, and Henry Ford discovered that a man could be used to turn one nut through one revolution, from day to day, to the last syllable of recorded time.

To industry, man became simply "a hand". To medicine he became "a case".

To parallel the mass production mammoths in industry, medicine developed what Zweig calls "the colossal department stores of human misery", the hospitals, in which doctors as though on moving belts flit from one "bed" to the next, "handling" one "case" after another, followed by a little crowd of gaping undergraduates. Sometimes they go down to the really high-speed production lines of the outpatient department, where a "case" moves from one specialist to another to have his various organ afflictions catalogued and dealt with.

But all illness revives infantile feelings of helplessness, of fear. Psychoanalysis has taught us how children symbolically incorporate or introject both "good" and "bad" objects. In illness, these bad objects turn out to be punishing objects, and only the good father or mother symbol can exorcise them. So, people do not like and do not understand the mechanization of healing. They seek for "natural" healers, for understanding of themselves as they are and feel. No amount of State legislation against quacks has ever suppressed astrologers, naturopaths, hypnotists, homœopaths, herbalists and faith healers.

The common man, however, is not the only rebel. Throughout the modern history of medicine rebels have arisen from the ranks of the learned doctors themselves.

Paracelsus, for instance, said that man was not a clock which you could dissemble into parts and assemble again, and he emphasized the will-to-health. During the nineteenth century there was an underground movement of romantics, who believed in *natura naturans*, the self-healing power of the organism whose will-to-health had been strengthened—Mesmer's magnetic theories were essentially based on this. They turned away from chemotherapy to Nature: water treatments, sun and air baths and the like. I should include Freud in this "underground" movement, for the analyst helps the patient to uncover deeply repressed memories, to perceive them in personal and social contexts of a real present, not of an imaginary, unreal past. As the patient's ego is strengthened, he no longer falls prey to the dark forces of infantile conflicts, of projected and introjected objects. Essentially, the patient cures himself.

In "scientific" medicine, the sick person is passive and is treated as an object. He does what he is told is good for the handling of his case. Religion, on the other hand, treats the person as subject, as an active agent, and so does psychotherapy. Consider the vast difference between the procedure in the consulting room of a heart specialist and that of a psychoanalyst—no apparatus, no pills, none of what Glover calls "the pugilistic methods of the psychiatrist".

Has the wheel of medicine then come full circle, from religion back to religion, through the purgatory of schizoid rationalistics? No, that other simile is better: the path of progress in science is spiral. We do return to our starting points, but at a higher level of insight, with more complex and sophisticated conceptualizations.

Consider again the case of the little girl who refused food. She was setting up a *circulus vitiosus*: the less she ate the more listless she became, and so would eat less. The parents and the doctor tried to treat her according to the prescriptions of their particular systems of roles, values and beliefs. In the case of the doctor, if I may repeat a phase from Glover, "absorption in the problems of organic pathology had blunted rather than sharpened psychological perception".

How was the vicious circle broken? In old-fashioned terms we might say by the loving-kindness of the parents.

In our modern terminology we spoke of social roles such as the authority of parents, of the distorted perceptions of the child, the lack of thinking in terms of social contexts displayed by the doctor. What had to be broken was a resonating system of roles inappropriate to the personality and psychological history of the patient; and it was broken by loving-kindness.

Experience often teaches wisdom. Science deepens insight by making wisdom more articulate and flexible. A comparable example would be to ask "how is a wound cured most quickly?", and, setting against the voice of experience —"by washing it clean"—the statement by a physiologist on the principles of asepsis and the circulation of biochemical and bacteriological agents in the blood. In other words, science gives us better or more appropriate means for reaching human goals.

The parents of the girl and the wife of the naval officer did not need to be told that love would aid the cures. What the experts did not tell them—because they were untrained in psychology—was in what particular ways their love should be expressed. Love often marches through the stomach; but that saw was useless and misleading in both these instances.

Similarly, just telling students that 50% of cases are psychological is like telling the mother to love her little daughter. What can they do about it?

The Training of a Psychologist.

"There is no psychological instruction worthy of the name in medical courses", to quote Glover (1949). Recently some fourth-year medical students during a tutorial discussion maintained that all psychology would ultimately be explainable in terms of the biochemistry and physiology of the brain and central nervous system. I gave them some examples, among them these: A personally maladjusted teacher is found to have more maladjusted children in his class than a stable teacher. Suppose in this instance the maladjusted teacher had an injection which straightened out his neurons. Would that settle the matter? Or would one also have to inject each maladjusted child? Each child might, of course, have to have a different injection, since the maladjustments are all different in their expression, and perhaps in their ætiology. Next, teachers have one of the most important functions in society, yet their status is low. How would one raise this by physiological means?

What these fourth-year students show is the extent to which the naïve materialism of the nineteenth century has affected their concepts about the doctor-patient relationship. "What cannot be put into a test tube does not exist."

Let us consider now the training of a psychologist in Melbourne.

Reflection on the history of medicine in particular, and of the practice of the sciences in general, led us to formulate policy somewhat as follows: To learn the languages and techniques of science requires practice on a reality level; therefore there must be more practical work than lectures.

Next, experimental psychology began in that same nineteenth century which, in medicine, saw the dismemberment of the person into a bundle of organs, destroyed the ancient art of healing and turned it into a collection of techniques for solving physiological puzzles. As I said in the first lecture, psychology did not begin by wanting to understand persons. It began with puzzles about "faculties", which are the analogues of organs. Having got out of that stage, it behoves us to teach the far more difficult concepts of measuring relations rather than entities, and to bring the students into active contact with human-beings-in-relation, so that they will not fall into the error of taking the sum of parts for the whole.

I assume that you will not misunderstand me as implying that the study of parts and the solving of puzzles is unnecessary. We must work harder than ever at puzzles, but in the end must try to put the pieces together, to treat them as at least part of a jigsaw, not a crossword.

The honours course in psychology, then, has two main aspects: the study of the relation of parts to wholes, and the study of the relations within and between wholes.

There is first an introductory year, Psychology Part I. This covers the fields of psychology and introduces the student to the elements of scientific method and to topics like learning, perception, emotion and motivation, the measurement of individual differences, and the elements of personality structure. Psychology Parts II and III deal with learning theory, behaviour theory and perception, the development of personality from infancy and finally the total integration of personality. These are called "the core course".

There are, in addition, three "specialist" courses. Collective behaviour is concerned with the effects of people on each other and with the structure and dynamics of social fields. Problems such as class differences, the origin of leadership and of prejudice, the kind and intensity of family relationships, are studied both in the laboratory and in the field. Psychopathology is concerned with the ætiology and pathology of intrapersonal disequilibrium. Applied Psychology (Industrial) is primarily a course in methods of assessment, the raw materials for which come from the socio-psychological systems of industry, and it includes vocational guidance and selection. Next year we

hope to have our own clinic in action, to train students while at the same time doing a community service.

The teaching process is built on syndicate work, tutorials and work periods. That is, the student in his first year begins to learn the elementary principles of working with and performing in small, cooperative groups. In his later years the syndicate methods are intensified. He learns from experience the difference between group goals and individual goals, and the effect of cooperation and competition on levels of aspiration and group equilibrium. That this learning process is more than superficial can be seen from the fact that teachers who have gone through this training are now successfully active in remodelling the teaching process in schools, to bring it into line with applied social psychology.

From his second year the student is brought into active contact with people in real social situations, not merely those of the laboratory. He learns to use the Binet and other methods of psychological examination and diagnosis. He is trained intensively in case history taking and in the theory and practice of interviewing. He has to go out and do field interviews in connexion with the collective behaviour research projects—last year the UNESCO tensions study, this year the Prahran City planning study—and he has to do case studies in psychopathology and vocational studies in applied psychology. The student has to stand on his own when he faces a subject in the field or the clinic, and when he is confronted by a final examination paper. The rest of his learning, however, is continuously subjected to the stimulus of his peer group's criticisms on the one hand and their active participation in laboratory and field work on the other.

The shortest way of summing up is to say that we attempt to create and to maintain a democratic matrix in which learning can, as far as feasible, grow untrammelled by interpersonal tension or by status tensions.

It will not have escaped you that the conceptual approach, as in medicine, is biologically oriented. We, too, think in terms of embryology, morphology, anatomy, and physiology of groups and social cultures. And we consider the implications of the concept of evolution.

One more stage is worth mentioning. A student who is a candidate for the master of arts degree is expected to range over the whole of human activity. Throughout the year there is a weekly seminar guided by representatives of other studies of human activity. Three-weekly periods are devoted to seminars on the methods, problems and concepts of general biology, anthropology, history, philosophy, political science, education, law and government, and finally language and the fine arts. These are, of course, additional to the major psychological studies and research required for the master of arts degree. By this we achieve two things: first, the student is related to other areas of human endeavour so that human values and motivations can be better understood; secondly, the Department of Psychology is related to other departments in the university through participation, and not merely at an administrative level.

The relations of the psychology department with other groups in our society are established through various activities—in education, social research, industrial research and vocational guidance.

You will have perceived by now that I regard psychology as a fundamental discipline, whose position in the social sciences is analogous to that of physics in the natural sciences and of physiology in the biological sciences.

If I have succeeded in convincing you, the implications for medical practice are clear. Like the psychologist, the doctor cannot divorce himself from social reality. Just as the psychologist can fail because he is so concerned with the dissection of psychological parts that he overlooks the wholeness of the personality, or is ignorant of the somatic factors in personality disturbance, so the physician can fail because he regards the patient as a case with a disease of an organ, overlooking its effects on other organs, or because he is ignorant of psychological and social factors in somatic disturbance.

But the doctor cannot be expected to spend another four years acquiring psychological techniques. How, then, can the doctor and the psychologist best cooperate?

Under the stress of war, the anxiety of physicians and psychiatrists at the rise of psychology as a profession was largely overcome. We might ask, why should there be anxiety?

I believe that two factors explain most of it. The first is that prejudice is a function of participation. The less one knows about a group, the easier it is to project hostility on to it. Physicians know very little about the present-day training and practice of psychologists. Those who do know seem free of this anxiety. Here in Melbourne, at any rate, cooperation between psychiatrists and psychologists is no longer a problem. In clinical practice, whether the psychiatrist or the psychologist shall carry out psychotherapy is determined by the needs and condition of the patient, not by reference to the sovereign rights of psychiatrists and psychologists. Such problems cease to be of importance where there is freedom of discussion and free access to knowledge. Even the question of lay analysis was authoritatively discussed and settled by Freud (1947) thirty years ago.

During their period of training in the psychiatric clinics of hospitals, psychologists learn much about the attitudes and special skills of psychiatrists. However, since psychiatrists are generally in charge of such clinics, it is necessary that they should know a good deal at first hand about psychological techniques. Here, too, a good beginning has been made. Up till 1946 the post-graduate diploma in psychological medicine at Melbourne allowed for ten hours' "instruction in 'normal' psychology". Since then the formal requirement has been stepped up to more than 375 hours spread over two years. And the demand for psychology in the medical undergraduate curriculum is rising, psychiatrists being especially insistent. Several former Beattie-Smith lecturers have emphasized this: Dr. H. M. Maudsley, Dr. Alan Stoller, Dr. Guy Springthorpe and Dr. A. J. M. Sinclair. Dr. Sinclair's paper in particular is well worth reading again for its emphasis on the need for introducing the medical student from his first week in the medical school to the study of "the whole man, not only a hypothetical somatic half".

The book edited by Witmer (1947) is worth mentioning here. It will give to those who are interested far more cases of the kind which the general physician meets so often in practice. In parentheses, let me urge all physicians to read this book, and express the hope that a similar fortnight's course of training will soon be tried in Melbourne.

A special aspect of the first factor—ignorance of the fields and methods of psychology—is the common notion that one knows by experience what is normal behaviour. This is a fallacy, or at any rate is true only as a very rough approximation. To establish adequate criteria for what is normal is one of the most difficult tasks in all biological sciences. First, personal experience is limited, and moreover limited largely to one's own class. There are class patterns of behaviour, and what is normal in one class is abnormal in another. Examples of interest to all medical men may be found in the Kinsey (Kinsey *et alii*, 1948) report on sex behaviour. (See also the references to Baker, 1950, Davis, 1943.) Secondly, one's view of what is normal is coloured not only by one's class prejudices but by one's own unconscious. An example of the former is the undue preference teachers show for the quiet, shy child who works hard; an example of the latter the utter lack of understanding and indeed hatred of a money-hungry business man for a spendthrift.

The second factor is the growing realization that mechanized medicine has destroyed not only the soul of the patient, but that of the physician as well. He no longer has the role of healer; and the desocialization of urban society is confronting him with problems to which the puzzle-techniques give no answer. There must be a close parallel in the ascending curves for "phenobarb and hypertension cases" and for divorce, for instance.[1] The physician, in other words, suffers a considerable degree of role-frustration. His anxieties would naturally be projected on

[1] See, for example, the articles by Dr. A. T. M. Wilson on family case work (Wilson, 1947), and on the prevention and treatment of marital problems (1949).

to psychologists who seem cheerfully to be dealing with whole persons in whole situations, whilst he lacks the theoretical background to help him in an integrated approach to the social personality.

Man is an organism whose needs are related to institutions, roles, human relations, internal psychological equilibria and biological factors. The general physician operates largely at the biological level, the psychiatrist at the level of individual emotional dynamics, the psychologists at the other three, the "social" levels. Yet what happens at one level affects all others. It is consequently quite meaningless to attempt a logical definition of the boundaries between these three professions. The notion of "national sovereignty" is out of date, and in science, certainly out of place. Where the labours of one end and of the other begin is a matter of practice, and in the face of our ever-increasing particulate knowledge, the best practice is teamwork.

This is not the place in which to put forward detailed proposals for the psychological training of physicians and psychiatrists. Social workers, who form the clinical team together with psychiatrists and psychologists, should of course be trained in psychology. Here in Melbourne I need not single them out, since the Diploma in Social Studies now requires three or four full psychology courses, depending on the special field in which the candidate will work. But from what I have said, the main lines should be clear: the medical student, whether he will become a psychiatrist or not, must learn about the development of the normal personality, and the forces that act on the person in social space. He must not merely learn the theory, but must experience the main methods of psychological measurement and diagnosis, the cognitive, emotional and social parameters of that behaviour which Lewin writes as "$B = f(P,E)$: behaviour is a function of personality and psychological environment. I have tried to indicate that P and E are themselves exceedingly complex functions. And finally, we may ask, why the medical undergraduate should be sheltered for three years from contact with people and their ways of life, and from his fourth year on be confined to "the department stores of human misery". Surely the methods used in training psychologists are general enough to be applicable also to the training of medical men. Surely the unbounded capacity for enthusiasm and hard work of the young men and women could be given greater scope by more contact with reality in social settings, and by the reorganization of teaching on syndicate lines.

CONCLUSION.

Let me attempt to sum up these lectures in broad sociological terms.

The development of the social sciences is accelerating, and through them we may achieve control over pathological social developments. In the social sciences, psychology occupies a key position.

There have been two revolutions in psychology stemming from the development of psychoanalysis and of social psychology. As a result, vast changes are impending in our knowledge of and therefore control over the forces that mould man's personality in society.

As personality is the product of social learning and social interaction, within a biological organism, so society is a web of interpersonal relations within an institutional, man-made structure—the body politic.

The concept of the individual which is still current in medicine and government, however, is abstract, juridical and mechanistic. In government it was meaningless, but no bar to good government—as then understood—in the days of feudalism, or in the days when physicians were mere wielders of scalpels and test tubes.

Now we know it to be not alone meaningless, but dangerous. For the State that operates with an abstract juridical concept of the individual is the authoritarian State in which government proceeds on the assumption that the State has a mind, a will, an ethic of its own, to which individuals are subordinate.

Similarly in medicine, the notion that the individual has a body that can be abstracted from the total social context in which he functions, from which he receives meaning and psychological structure and through which he expresses his needs, implies that signs and symptoms are being confused, and that machines are being used to the exclusion of insight.

The rise in the incidence of psychosomatic disorders and the spread of mental epidemics are at once a reflection of the disorganized state of society, and a challenge to the ancient and honourable calling of the healer.

REFERENCES.

Alschuler, R. H., and Hattwick, L. W. (1947), "Painting and Personality", London, Cambridge University Press.

Asch, S. E. (1945), "Forming Impressions of Personality", *The Journal of Abnormal and Social Psychology*, Volume XLI, page 258.

Baker, S. J. (1950), "Sexual Jealousy and Democracy", *International Journal of Sexology*, May, page 1.

Bion, W. R. (1948), "Experiences in Groups", *Human Relations*, Volume I, pages 314 and 487.

——— (1949), "Experiences in Groups", *ibidem*, Volume II, pages 13 and 295.

Bruner, J. S., and Goodman, C. C. (1947), "Values and Need as Organizing Factors in Perception", *The Journal of Abnormal and Social Psychology*, Volume XLII, page 33.

Curle, A., and Trist, E. L. (1947), "Transitional Communities and Social Re-connection", *Human Relations*, Volume I, pages 42 and 258.

Davis, A. (1943), "Child Training and Social Class", in "Child Behavior and Development", by Barker, Kounin and Wright. New York, McGraw-Hill, page 607.

Dicks, H. V. (1947), "Clinical Studies in Psychopathology", London, Edward Arnold, Second Edition.

——— (1950), "Personality Traits and National Socialist Ideology", *Human Relations*, Volume III, page 111.

Fenichel, O. (1946), "The Psychoanalytic Theory of Neurosis", London, Kegan Paul.

Freud, S. "The Future of an Illusion", London, Hogarth Press.

——— (1947), "The Question of Lay Analysis", London, Imago.

Fromm, E. (1941), "The Fear of Freedom", New York, Rinehart (London, Routledge, 1942).

Glover, E. (1949), "Psychoanalysis", London, Staples Press, Second Edition, page 328.

Goldstein, K. (1939), "The Organism", New York, American Book Company.

Halstead, W. (1947), "Brain and Intelligence", University of Chicago Press.

Harrower, M. R. (editor) (1947), "Training in Clinical Psychology", New York, Josiah Macy, Junior, Foundation.

Harvard Report, The (1948), "The Place of Psychology in an Ideal University", Report of the University Commission to Advise on the Future of Psychology at Harvard, London, Geoffrey Cumberlege.

Homburger, E. (1947), "Dramatic Productions Test", in Murray, H. A., "Explorations in Personality", page 552.

Jones, E. (1949), "Hamlet and Oedipus", London, Gollancz.

Kinsey, A. C., Pomeroy, W. B., and Martin, C. E. (1948), "Sexual Behaviour in the Human Male", Philadelphia and London, W. B. Saunders.

Klopfer, B., and Kelley, D. M. (1942), "The Rorschach Technique", New York, World Book Company.

Lashley, K. (1929), "Brain Mechanisms and Intelligence", University of Chicago Press.

——— (1933), "Integrative Functions of the Cerebral Cortex", *Physiological Reviews*, Volume XIII, page 1.

Lewin, K. (1947), "Frontiers in Group Dynamics. Concept, Method and Reality in Social Science: Social Equilibria and Social Change", *Human Relations*, Volume I, page 1.

Linton, R. (1945), "The Cultural Background of Personality", New York, D. Appleton Century Company.

Murray, H. A. (1947), "Explorations in Personality", New York, Oxford University Press.

Newcomb, T. M. (1947), "Autistic Hostility and Social Reality", *Human Relations*, Volume I, page 69.

Newman, P. H. (1944), "The Prisoner of War Mentality", *British Medical Journal*, Volume I, page 8.

Richards, T. W. (1946), "Modern Clinical Psychology", London, McGraw Hill.

Schafer, R. (1948), "The Clinical Applications of Psychological Tests", New York, International Universities Press.

Schneirla, T. C. (1946), "Problems in the Biopsychology of Social Organisation", *The Journal of Abnormal and Social Psychology*, Volume XLI, page 385.

Sinclair, A. J. M. (1945), The Beattie-Smith Lectures, "The Psychological Reactions of Soldiers", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, pages 229 and 261.

Slavson, S. R. (1943), "An Introduction to Group Therapy", New York, Commonwealth Fund.

Snyder, W. V. (1947), "The Present Status of Psychotherapeutic Counselling", *Psychological Bulletin*, Volume XLIV, page 297.

Stoller, A. (1948), The Beattie-Smith Lectures, "Social Health and Psychiatric Service", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, pages 1 and 29.

Sutherland, J. D. (1950), "Clinical Psychology", *Universities Quarterly*, Volume IV, page 157.

Thompson, D'A. W. (1940), "Science and the Classics", Oxford University Press.

Wilson, A. T. M. (1947), "The Development of a Scientific Basis in Family Case Work", *Social Work*, Volume IV, page 62.

——— (1949), "Some Reflections and Suggestions on the Treatment of Marital Problems", *Human Relations*, Volume II, page 233.

Witmer, H. L. (editor) (1947), "Teaching Psychotherapeutic Medicine", New York, The Commonwealth Fund.

Zweig, S. (1931), *"Die Heilung durch den Geist"*, Leipzig, Insel-Verlag.

THE EFFECTS OF SODIUM CITRATE IN LEAD POISONING AND LEAD ABSORPTION: LEAD POISONING.[1]

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IN the treatment of lead poisoning a substance which would allay the acute symptoms, increase the excretion of lead without a concomitant exacerbation of symptoms and be simple to administer would be very valuable. Sodium citrate fulfils these requirements to a high degree.

Treatment of lead colic by intravenous injections of calcium chloride or calcium gluconate or sodium thiosulphate is very successful. Subsequent to the intravenous injection of the calcium salt, a diet rich in calcium and calcium medication by mouth are frequently used, with the object of storing the lead in the bones. Such procedure is open to the objection that the lead is not got rid of and that symptoms may, and frequently do, recur on cessation of the treatment. Recurrence of symptoms subsequent to adequate treatment by sodium thiosulphate is rare. In order to get rid of the stored lead various methods have been adopted, such as the oral administration of ammonium chloride, potassium iodide, sodium bicarbonate or sodium acid phosphate, and the use of a diet of low calcium content.

Such "deleading" may lead to recurrence of symptoms, even in severe form, unless carefully controlled. Moreover, some of these substances are not pleasant to take. Methods requiring intravenous injections are naturally not so convenient as oral methods. Kety and Letonoff (1943) first showed the value of the sodium citrate treatment in cases of lead poisoning. Kety (1942) had previously shown that sodium citrate in aqueous solution produced with lead salts a complex negative ion, containing lead of very low dissociation. If the physiological effects of lead salts are due to the positive lead ion, then it was reasonable to suppose that, if the concentration of this positive lead ion could be reduced by its conversion to a complex negative ion of low dissociation, the physiological effects of a certain total amount of lead would be decreased. Letonoff and Kety (1943) found that there was a significant increase in the total daily lead excretion in urine and fæces, and Kety and Letonoff (1943) that there was a decrease in the concentration of lead in the blood. Although in eight out of nine cases there was an increase in the daily excretion of lead in urine and fæces, considered separately, such increases were not statistically significant, although approaching significance. Symptoms rapidly decreased during the treatment. There were no consistent alterations in the calcium, inorganic phosphate, protein, chlorine or carbon dioxide contents of the blood.

[1] A subsequent paper will deal with the effects of sodium citrate in lead absorption.

It was thought that such a promising method of treatment justified further investigation. This paper describes the results obtained in the oral treatment by sodium citrate of a number of patients suffering from lead poisoning. A subsequent paper will describe the results obtained from its use as a prophylactic agent by a considerable number of persons who were exposed to a lead hazard in an accumulator factory. In the assessment of the therapeutic value of sodium citrate for the purposes under discussion, the criteria considered were the feeling of well-being or otherwise of the subjects, the effects on symptoms (such as colic, abdominal pain, muscular and joint pains, nausea, loss of appetite, loss of weight, loss of muscular strength, fatigue, headache *et cetera*), and the results of the following laboratory tests: the stippled cell count per million red cells, the ratio of large lymphocytes and monocytes to small lymphocytes, the concentration of lead in the urine, and in some cases the concentration of lead in the blood.

In ten cases of lead poisoning blood films were examined before treatment commenced and approximately weekly thereafter; urinary lead concentrations were determined before the treatment and on samples collected every second day during the course of the treatment. The patient was directed to collect approximately equal quantities on three occasions during the day—morning, afternoon and evening—so that the sample was representative of the day's output. There were some slight variations in certain cases in the frequency of the urinary examinations.

The dose of sodium citrate was four or five grammes in an ounce or two of water three times per day.[1]

The method of analysis of the urine for lead in the majority of cases was that of Taylor (1925), slightly modified by Shiels (1936). In this method ammonium oxalate and calcium chloride are added to the urine previously made slightly acid with acetic acid. The lead is precipitated with or adsorbed on the calcium oxalate precipitate. For Cases I and IA the lead concentrations were determined by two methods for the first 13 of the determinations. One method was that referred to above; the other was a short dithizone method described by Shiels (1938), which requires only three-quarters of an hour for a determination. That the results obtained by the two methods agree sufficiently well is shown in Table I. The other 23 determinations made in the following-up of this patient were carried out by the short dithizone method of Shiels (1938).

Effects on Urinary Lead Excretion.

The patients considered in Part I had all ceased work and industrial lead absorption had stopped. Within a day or two of their commencing the citrate treatment the concentration of lead in the urine had increased. That this increased concentration represented an increased elimination of lead was shown by the facts that the citrate therapy did not cause any decrease in the daily output of urine, and that the concentration expressed as weight (microgrammes) of lead per gramme of solids excreted in the urine was also increased.

The concentrations in microgrammes of lead per gramme of solids in the urine were determined from the following formula: $U_s = \frac{3{\cdot}8 \times U}{G}$ where U_s = weight in microgrammes per gramme of solids, U = urinary lead concentration in microgrammes per 100 millilitres, and G represents the specific gravity (*S.G.* 1026, $G = 26$).

The urinary lead concentration before and after the commencement of citrate therapy is shown in Tables II[2] and III.

The mean increase was 0·07 milligramme per litre, and the standard error was 0·0202. The t value was 3·465 (Fisher, 1936). For nine determinations a t value of 3·25

[1] Analysis of the sodium citrate (British Pharmacopœia) showed that in the doses taken daily it could not contribute any appreciable amount to the urinary lead concentration even if all the lead it contained were excreted in the urine.

[2] Case I was investigated fully, the results being considered in detail later. "Case I(a)" refers to the same person as "Case I". The results for Case I(a) refer to resumption of citrate treatment after an interval of twenty-two days without it.

corresponds to a P value of 0·01. The P value corresponding to a given value of t represents the likelihood that that value of t could be exceeded by chance. For significance a P value of 0·05 would be required. The above increases then show a significant rise due to the citrate.

Through a misunderstanding one patient (Case V) was receiving a calcium-rich diet for a week while taking the citrate. He showed a slight decrease in urinary lead concentration after a few days on the citrate. Subsequently the calcium-rich diet was stopped, on July 9, 1947. On July 10 his urinary lead excretion was 0·08 milligramme per litre. On July 12 it had risen to 0·15 milligramme per litre, and on July 21 it was still up to 0·12.

TABLE I.

Urinary Lead Concentrations.

By Modified Taylor's Method. (Milligrammes per Litre.)	By Short Dithizone Method of Shiels. (Milligrammes per Litre.)
0·386	0·390
0·33	0·363[1]
0·387	0·363
0·393	0·388
0·401	0·405
0·420	0·425
0·43	0·420
0·34	0·320
0·343	0·343
0·24	0·265
0·34	0·363
0·33	0·32
0·35	0·333
Mean 0·359	0·361

[1] Citrate therapy commenced after this sample was taken.

If the values 0·08 and 0·15 milligramme per litre had been used in Table I instead of the values obtained when the treatment was complicated by the calcium-rich diet, an even more significant rise in the urinary lead values would have been shown in the group. Thus the mean increase would become 0·079 milligramme per litre and the standard error 0·0178, giving a t value of 4·43.

TABLE II.

Concentration of Lead in the Urine Before and After Commencement of Citrate Therapy.

Case.	Before. (Milligrammes per Litre.)	After (1 to 2 Days). (Milligrammes per Litre.)	Increase. (Milligrammes per Litre.)	Patient's Occupation.
I	0·36	0·38	0·02	Grinding materials such as white lead.
IA	0·10	0·33	0·23	Grinding materials such as white lead.
II	0·08	0·16	0·08	Storage battery work.
III	0·13	0·24	0·11	Keeping an orchard.
IV	0·19	0·25	0·06	Making lead arsenate.
V	0·18	0·16	-0·02	Lead smelting.
VI	0·02	0·07	0·05	Plumbing.
VII	0·14	0·23	0·09	Storage battery work.
VIII	0·08	0·11	0·03	Storage battery work.
IX	0·08	0·13	0·05	Making lead arsenate.
Mean ..	0·136	0·206	0·07	

For $n = 9$, a t value of 3·25 has a corresponding P value of 0·01, and a t value of 4·781 a P value of 0·001. The value of t found—namely, 4·43—would correspond to a P value much less than 0·01, approaching 0·001.

In two cases the maximum values (greater than those shown in the table) were not reached until five or six days after commencement of the citrate therapy. In two cases (Cases I and IA) the maximum was reached at twenty-nine days after citrate therapy commenced and three days after its resumption. With the exception of Cases I and VII, the values had returned to the normal range in from ten to twenty days after the citrate therapy commenced.

The mean increase was 1·42 and the standard error 0·295, giving a t value of 4·8.

TABLE III.

Increase in Urinary Lead Concentrations Expressed in Microgrammes of Lead per Gramme of Total Solids in the Urine.

Case.	Before Commencement.	After Commencement.	Increase.
I	4·8	4·9	0·1
IA	1·3	3·7	2·4
II	2·7	3·3	0·6
III	2·9	4·0	1·1
IV	4·3	6·5	2·2
VI	1·2	3·0	1·8
VII	4·5	5·3	0·8
VIII	1·9	4·8	2·9
IX	1·9	2·8	0·9
Mean ..	2·83	4·25	1·42

For $n = 8$ and $t = 3·355$, the P value is 0·01. For $t = 5·041$, the P value is 0·001 (Fisher and Yates). The above t value of 4·8 is therefore highly significant.

Data for expressing the concentration in Case V in the above way were not available.

Figure I shows graphically the urinary lead concentration in relation to the time which had elapsed since the treatment commenced. On the ordinate at 0 are plotted the values of the urinary lead concentration in milligrammes per litre just prior to the commencement of the citrate therapy. The abscissæ are the times in days since the therapy started. It is clear that there was in all cases a substantial rise in the urinary lead concentration. The increased values are maintained for varying times. In the majority of cases the values five days after treatment started were still well above the initial values.

TABLE IV.

Ratio of Large Lymphocytes plus Monocytes to Small Lymphocytes.

Case.	Before Commencement of Therapy.	After Commencement of Therapy.[1]	Increase in Ratio.
I	1·5	5·6 (18)	4·1
IA	2·2	4·1 (7)	1·9
II	2·0	3·2 (6)	1·2
III	1·4	4·4 (8)	3·0
IV	1·6	3·0 (16)	1·4
V	1·6	2·4 (6)	0·8
VI	5·5	8·0 (3)	2·5
VII	1·0	3·5 (22)	2·5
VIII	1·4	7·7 (14)	6·3
Mean ..	2·02	4·65	2·63

[1] The figures in parentheses show the number of days after the commencement of citrate therapy when the ratio reached the value shown.

Effects on the Ratio of Large Lymphocytes Plus Monocytes to Small Lymphocytes.

Influence of citrate therapy on the ratio of large lymphocytes *plus* monocytes to small lymphocytes is shown in Table IV. The mean increase was 2·63.

The standard error was 0·522, giving a t value of 5·04. For $n = 8$ and $t = 5·041$, the corresponding value of P is 0·001. Thus the increase was highly significant. It has already been shown (Shiels, 1937) that the ratio of large

lymphocytes *plus* monocytes to small lymphocytes is closely related to the clinical condition, much more closely than the stippled cell count in fact. The increase in this ratio to well above 2·0 was accompanied by a pronounced improvement in general well-being and disappearance of symptoms.

When the ratio was estimated at a fixed time after commencement of the citrate therapy (say ten days), it was found that in some cases the ratio had not yet reached its highest value, and in other cases it had reached its highest value and was falling again. Even in these circumstances there was still a highly significant increase in the ratio above its values before the commencement of the citrate therapy. This is shown in Table V. The mean increase was 1·60.

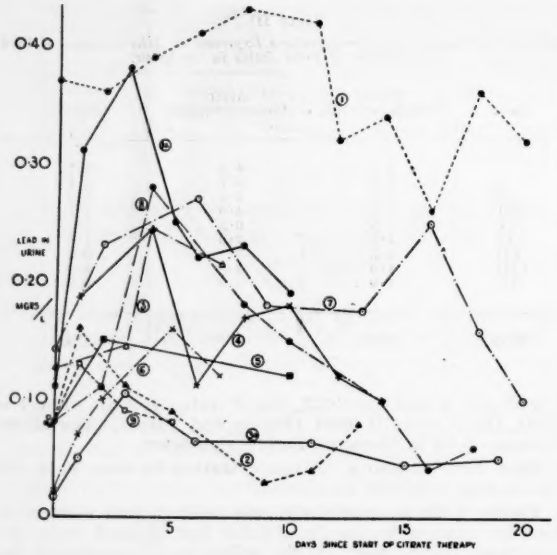

FIGURE I.

TABLE V.

Ratio of Large Lymphocytes plus Monocytes to Small Lymphocytes.

Case.	Before Commencement.	Ten Days After Commencement.	Increase in Ratio.
I	1·5	3·3	1·8
IA	2·2	3·6	1·4
II	2·0	3·2	1·2
III	1·1	1·4	0·3
IIIA	1·5	5·0	3·5
IV	1·6	2·6	1·0
V	1·6	1·9	0·3
VI	5·5	6·0	0·5
VII	1·0	2·1	1·2
VIII	1·4	6·0	4·6
X	1·1	2·9	1·8
XA	2·0	4·0	2·0
Mean ...	1·9	3·5	1·6

The standard error was 0·355 and the t value was 4·507. For $n = 11$ and $t = 4·337$, the P value is 0·001. Thus there was at this time interval a highly significant increase in the ratio.

The effects on the ratio in relation to the time since the commencement of the citrate therapy are shown graphically in Figure II. It is seen that in all cases there was a substantial rise in the ratio shortly after the taking of citrate commenced. The ratio continued to increase for some days, eventually falling (in the cases in which the tests were continued long enough) to about the values which are found in non-exposed persons.

Effects of Citrate Therapy on Stippled Cell Counts.

The stippled counts were done by the light fixed technique described in a previous paper (Shiels, 1937) but using magnification × 900.

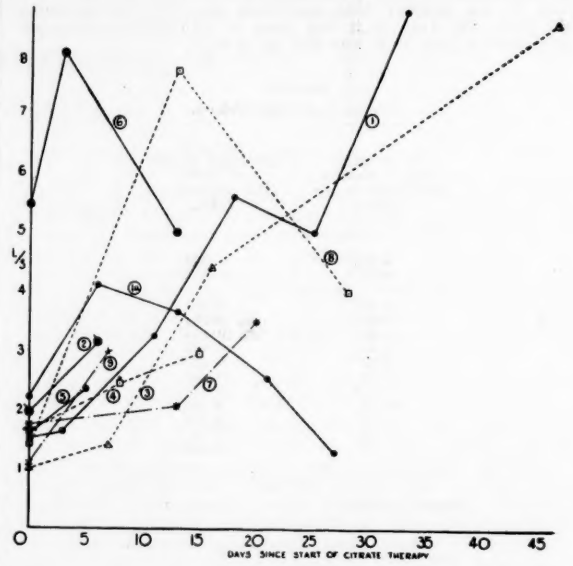

FIGURE II.

The effects on stippling were not consistent in the earlier stages of the treatment. In some cases the stippled cell counts increased during the early stages of the citrate therapy, but in the majority there was a rapid fall in the numbers. The rise in some of the counts is not evidence against the beneficial effects of the citrate, since it is well known that in cases of lead poisoning increases in the stippled cell count not infrequently accompany an improvement in clinical condition. Eventually, in times varying from about two weeks to two months, the counts returned to normal. The results are shown in Table VI.

Effects of Citrate Therapy on Symptoms.

The effects on the symptoms were prompt, improvement being noticed in some cases as early as the day following the commencement of the treatment. In some cases the colic had been removed by the intravenous administration of calcium gluconate before the citrate therapy started. In some cases the citrate was the only treatment given. It promptly relieved abdominal pain, nausea disappeared, appetite improved, muscular strength improved, muscular pain diminished, and the general feeling of well-being was vastly improved.

Reports of Cases.

Case I is of special interest owing to the length of time during which the high urinary lead concentration was maintained. It shows clearly the pronounced fall in urinary lead concentration on cessation of the citrate therapy and its pronounced rise on resumption of the therapy, and the final fall as the quantity of available lead finally decreased. The results are shown graphically in Figure III. This case also shows clearly the complete transition of the ratio from that of a lead-poisoned person (which is approximately the same as that of persons not exposed to significant amounts of lead) through the earlier phase of recovery, when relatively high values are found, to the final stage of recovery, when the value returns to that of a normal non-exposed person.

TABLE VI.

Stippled Cells per Million Red Cells.

Case.	Before Commencement.				After Commencement.			
	Coarsely Stippled.	Medium Stippled.	Finely Stippled.	Total.	Coarsely Stippled.	Medium Stippled.	Finely Stippled.	Total.[1]
I	14,300	12,500	23,000	49,800	10,600	14,200	21,900	46,700 (6)
					3800	3200	8600	15,600 (25)
Ia	2300	2800	19,900	25,000	2600	4300	6800	13,700 (6)
					0	200	2100	2300 (34)
II	1600		4200	5800	3400		4300	7700 (6)
III	11,600	4000	4400	20,000	6200	1300	4000	11,500 (6)
					500	200	2200	2900 (47)
IV				13,500				5500 (8)
								2500 (20)
V	760	2300	7600	10,600 (one day after)	600	2300	17,100	20,000 (6)
					0	1800	12,000	13,800 (11)
					0	400	1900	2300 (68)
VI	0	300	8200	8500	230	1400	4200	5830 (3)
					0	0	4800	4800 (13)
VII	6600		8400	15,000	13,100		19,100	32,200 (6)
					600		4200	4800 (31)
VIII	1430	1070	11,800	14,300	940	470	11,100	12,500 (13)
					0	200	9000	9200 (28)

[1]The figures in parentheses indicate the number of days after the commencement of citrate therapy at which the tests were made.

Each urine sample represented an average of the daily output. The patient was instructed to fill the bottle (about 700 millilitres) by passing an approximately equal amount directly into it on three occasions during the day. Fresh samples were collected at intervals of two days. Two curves are shown in Figure III for the urinary lead concentrations, one for which the concentrations are expressed in milligrammes per litre, and one in which they are expressed in microgrammes of lead per gramme of solids in the urine. It will be seen that, with occasional slight variation, they follow the same general course. Since this was not a hospital patient, there was unfortunately no opportunity of following the fæcal lead excretion.

CASE I.—This patient (whose findings are recorded in Figure I) had been engaged in the grinding of materials containing lead such as white lead. Three or four months before being examined by his doctor, he had noticed mild malaise which gradually grew worse. For a week previous to being examined, he had suffered from abdominal colic. Conjunctival and oral pallor, blue line, some facial pallor, and loss of subcutaneous fat in the face were apparent, and besides the colic he complained of loss of appetite, nausea and lassitude, being readily tired and disinclined even for short walks. He was referred to this division, having given up his work, and was first examined on March 31, 1947, when the stippled cell count was 49,800 per 1,000,000 red cells, his cell ratio 1·49,[1] and his urinary lead content 0·45 milligramme per litre; the hæmoglobin value was 65% to 70% (Tallquist). Examination of blood films revealed pronounced anisocytosis and poikilocytosis. Treatment with citrate was started on April 3, his urinary lead excretion on this day (prior to the taking of citrate) being 0·36 milligramme per litre. While he was receiving citrate treatment his urinary lead excretion rose to a maximum of 0·50 milligramme per litre, averaging 0·36 milligramme per litre (taken every second day) for thirty-one days. By April 9 he felt much better, was much more active and was going for walks, and his appetite was much better. He said that he had enjoyed his first "square meal" for months. By April 21 he was feeling better still, and his appetite was good, but he still tired rather easily. Citrate treatment was stopped on May 5, after thirty-one days. After twenty-one days without citrate the stippled cell count had risen from 15,000 to 25,000 per 1,000,000 red cells and his cell ratio had fallen from 8·7 to 2·2. The urinary lead excretion had fallen from 0·35 to 0·11 milligramme per litre and averaged 0·142 milligramme per litre for seventeen days without citrate. On May 12 he reported that his appetite was not so good, but otherwise he was about the same. However, he did not look so well. Citrate therapy was resumed on May 27. The urinary lead excretion had risen to 0·31 milligramme per litre by May 28, and averaged 0·26 milligramme per litre for the next ten days. On June 2 the cell ratio had risen again to 4·1 and the stippled cell count had fallen to 13,800 per 1,000,000 red cells. The hæmoglobin value was 80% to 85% (Tallquist). Examination of a blood film revealed less anisocytosis and very slight poikilocytosis. He now felt better again. His urinary lead excretion subsequently fell fairly rapidly, until on June 29 the amount was 0·075 milligramme per litre. This fall was accompanied by a drop in the cell ratio to 1·3 on June 23, when the stippled cell count was 4580 per 1,000,000 red cells, and there was no coarse stippling. The hæmoglobin value was then 85%. His cell ratio at that stage had fallen to a value in the range characteristic of persons not exposed to significant amounts of lead.

Treatment ceased on June 29. On June 30 the stippled cell count was 2200 per 1,000,000 red cells, and there was no coarse stippling. The cell ratio was 1·67. The urinary lead excretion was 0·029 milligramme per litre on July 4. He could then be regarded as having recovered.

CASE III.—The patient was an elderly orchardist, who was poisoned by lead arsenate. He was admitted to a country hospital on February 5, 1948. He was an Italian with hardly any knowledge of English, and an industrial history was difficult to obtain. Apparently he himself had not used any lead arsenate during the last spraying season, but his son had. On his admission to hospital he had abdominal colic, constipation, bilateral wrist drop, and almost complete bilateral deltoid paralysis; his appetite was poor, and he was anæmic and very pale.

On February 7 he was put on a calcium-rich diet for five days, and then on eliminative treatment with sodium acid phosphate for five days. He was then put on a calcium-rich diet for three days, and was then examined by this division. At that time he had improved considerably, in that the colic and constipation had gone, but pronounced weakness in both deltoids was still present. He was having liver extract for anæmia. He commenced sodium citrate therapy on February 20. On that day his stippled cell count was 20,000 per million red cells (11,000 coarsely, 4100 medium and 4400 finely stippled); there were also 3000 polychromasic cells. The cell ratio was 1·05 and the urinary lead excretion was 0·128 milligramme per litre. Examination of blood films revealed considerable anisocytosis, some poikilocytosis and hypochromic staining. On February 23 the urinary lead excretion had risen to 0·143 milligramme per litre, and the lead content of the blood was 0·107 milligramme per 100 grammes of blood. On February 24 the urinary lead excretion was 0·25 milligramme per litre. On February 25 the lead concentration in his blood was 0·074 milligramme per 100 grammes. On February 27 he was transferred to the Royal Melbourne Hospital, when the stippled cell count was found to have fallen to 11,500 per 1,000,000 red cells and the cell ratio to have risen to 1·4. The urinary lead excretion on that day was 0·185 milligramme per litre and the lead content of the blood 0·085 milligramme per 100 grammes. He was off citrate treatment for several days, and the urinary lead concentration fell to 0·017 milligramme per litre on March 3 and 4. Citrate therapy was resumed after this sample had been taken, and on March 5 the urinary lead excretion had risen again to 0·05 and on March 7 to 0·11 milligramme per litre. On March 8 the stippled cell count was 8900 per 1,000,000 red cells (2700 coarsely, 1200 medium and 5000 finely stippled cells), and the cell ratio had risen to 4·4. He now felt much better; his deltoid muscles and the extensors of the forearm and also his grip were much stronger. His appetite was good. On March 19 his

[1] "Cell ratio" throughout these reports refers to the ratio of large lymphocytes *plus* monocytes to small lymphocytes.

urinary lead excretion was 0·05 milligramme per litre. On March 22 he ceased taking citrate and was given calcium. On April 8 his stippled cell count was 2850 per 1,000,000 red cells (620 coarsely stippled and medium), and his cell ratio was 8·5. On April 9 and 10 his urinary lead excretion was 0·06 milligramme per litre. He was discharged from hospital on April 24.

Some samples of urine were tested for arsenic, the first being examined on March 3 and 4, when the concentration of arsenic was 0·10 milligramme per litre. This was before the resumption of citrate therapy. The concentration of arsenic on March 7, after three days of citrate therapy, was 0·18 milligramme per litre. On March 15, 19 and 23 respectively it was 0·05, 0·03 and 0·02 milligramme per litre. It is possible that the citrate increased the elimination of arsenic.

The results in this case are shown in Figure IV.

CASE VI.—Case VI is of some interest, in that the major complaint was of severe pain in the forearms in attempting to flex the arm or extend the wrist. The patient was a plumber, who had had almost twenty years at the trade, but had never noticed any symptoms of lead poisoning. Recently he had been engaged on a good deal of heavy lifting of materials, much more than was normally associated with his ordinary work as a plumber. When he was first examined on July 27, 1949, his stippled cell count was 6300 per 1,000,000 (coarsely stippled nil, medium stippled nil, fine 6300). His cell ratio was 1·59. He stopped work on that day. On August 2 his urinary lead excretion was 0·02 milligramme per litre, the cell ratio was 5·46 and the stippled cell count was 8500 per 1,000,000 red cells. There was no constipation, and his appetite was satisfactory. He said that he had had some chest pain some weeks earlier which had cleared up after he had taken powders (probably alkali powder for indigestion) prescribed by his doctor. He was a cigarette smoker and rolled his own cigarettes.

Almost the only indication that his symptoms might have been due to lead was the nature of his occupation and the cell ratio, with some slight indication, perhaps, from the finely stippled count. However, as a trial, citrate therapy was tried, starting on August 2. On August 3 the urinary lead concentration had risen to 0·07 milligramme per litre. On August 5 his stippled cell count was 5800 per 1,000,000 red cells (230 coarsely, 1400 medium and 4200 finely), his cell ratio was 8·0 and his urinary lead excretion was 0·10 milligramme per litre. On that day he said that he felt much better, and "full of vim". His right arm was nearly better, there being no pain on effort and plenty of power. He felt so well and vigorous that the medicine was described as "magic". On August 7 his urinary lead excretion was 0·16 milligramme per litre and on August 9 it was 0·12 milligramme. On August 15 his stippled cell count was 4800 per 1,000,000 red cells (all finely stippled) and his cell ratio was 5·0. He did not report to this division for any further investigation.

The results of this case are shown in Figure V.

CASE VIII.—This patient was a battery worker, who complained of abdominal pain, poor appetite, loss of weight, stiffness in the knees and back, and great tiredness at the end of the day's work. He was first examined by this division on December 1, 1948. He was recommended to his doctor for citrate treatment, but did not actually start the treatment until December 8, the intervening time being spent away from lead hazard. The findings in this case are shown in Figure VI.

The other cases presented no special features, but were certainly cases of lead poisoning with symptoms of colic, abdominal pain, nausea, vomiting and loss of appetite.

The results of the tests in these cases followed generally similar curves to those shown in Figures III, IV, V and VI.

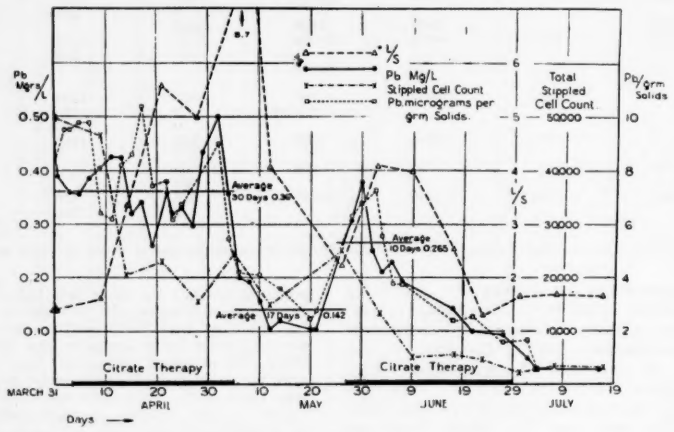

FIGURE III.

Discussion.

In assessing the value of the method of treatment, the criteria considered were the effect on the symptoms and signs, the general feeling of good or ill health, the effect on lead excretion or on the activity of lead contained in the tissues, and the effects on any useful diagnostic laboratory tests.

In regard to the first point, it has already been shown that the commencement of the sodium citrate treatment was followed in all cases by prompt relief of symptoms and improvement in general well-being. In none of the cases considered was severe abdominal colic present at the time the citrate treatment commenced. In some cases it had been present, but had been relieved by intravenous calcium gluconate therapy or other means. In the majority of cases abdominal pain was present at the time when the citrate therapy was started.

Kety and Letonoff (1943) have shown that sodium citrate given intravenously dramatically relieved abdominal colic in two out of four cases.

In regard to the second point, it has been shown that there was a significant increase in the urinary lead con-

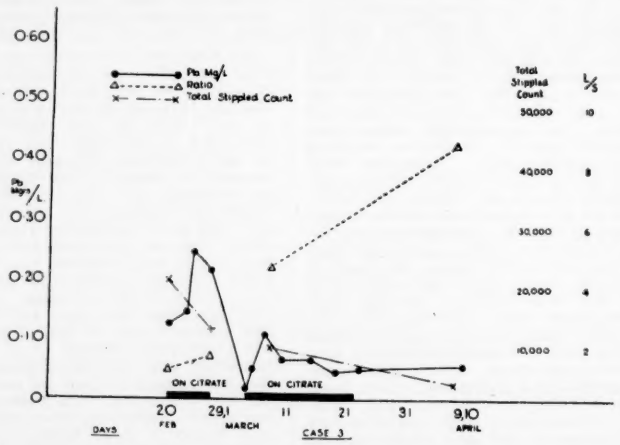

FIGURE IV.

centrations after the citrate therapy began, whether the concentrations were expressed as milligrammes of lead per litre, or as microgrammes of lead per gramme of solids in the urine. Although the daily output of urine was not measured (most of the patients not being hospital inmates), the impression was gained that the citrate therapy caused an increase in the daily output, as would be expected. It appears certain, then, that the citrate caused a real increase

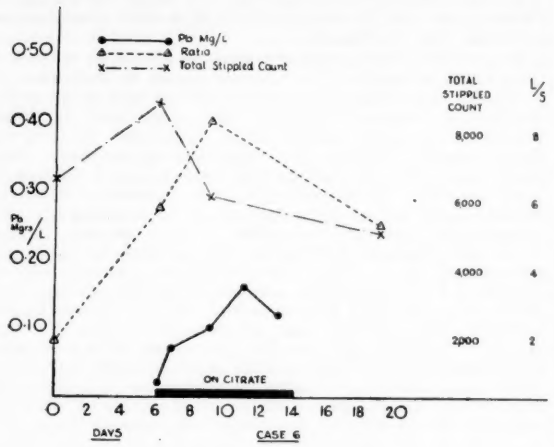

FIGURE V.

in the amount of lead excreted in the urine. This increase was not accompanied by any exacerbation of symptoms, as sometimes occurs with other "deleading" agents.

Kety and Letonoff (1943) and Letonoff and Kety (1943) have shown in 16 cases that there was a significant decrease in the lead concentration in the blood at four days after citrate therapy began. Subsequently to this decrease, and

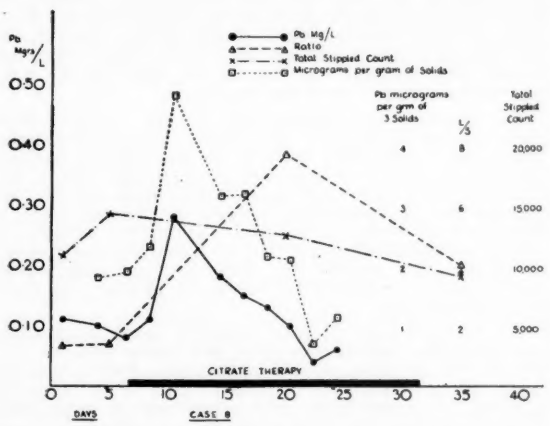

FIGURE VI.

while still under citrate treatment, in four of these 16 cases there were temporary increases in the blood lead concentration to values greater than or close to the respective values found before the citrate therapy began. Of these four cases, in two the increase was to values greater than, in one to a value equal to, and in one to a value slightly less than, the respective pre-treatment values. In none of these cases was there a return of symptoms during the increases in blood lead concentration.

In the present investigation it was found that at four days and after from the beginning of citrate therapy there was a significant increase in urinary lead excretion. Actually there was a significant increase in one or two days, but the results of the fourth day have been used in this discussion for comparison with Kety's and Letonoff's results.

These facts point to the conclusion that the taking of citrate causes the lead or a considerable fraction of it to be in a form which is more easily excreted by the kidney, and which is less active physiologically in the blood, than the form in which it normally occurs in the absence of citrate treatment.

These conclusions agree with those put forward by Kety and Letonoff (1943).

In regard to the cell ratio, it has been shown previously (Shiels, 1937, 1950) that the cell ratio was the most useful laboratory indication of the clinical condition of the patient and that in cases in which lead poisoning is present at the time of the examination the ratio in the very great majority of instances will be below 2·0.

During recovery the value rises. When it has reached or exceeded the value 2·0, symptoms have usually disappeared (with the exception of joint pains, which may sometimes persist long after the patient feels well in all other respects). Paralysis, of course, may persist for a considerable time after.

Ultimately, when the circulating lead has been reduced to normal values (or rendered inactive), the ratio will have fallen to within the range of values found in non-exposed persons.

In the present investigation it has been shown that citrate therapy causes a prompt increase in the ratio to well above 2·0, and that such increase is accompanied by a disappearance of symptoms.

In three instances the ratio before citrate treatment began was 2·0 or over. The reasons for this in Cases Ia and VI will be clear from the clinical histories. In Case II a certain amount of recovery had taken place before this test was performed and before the citrate treatment was begun.

It is, of course, admitted that mere removal of the patient from lead exposure will result in abatement of symptoms and an increase in the cell ratio; but the experience of the senior author (D.O.S.) with a large number of cases of lead poisoning indicates that in the absence of treatment such changes would not be so pronounced or so rapid as they were under citrate therapy.

Again, mere removal from lead exposure would not cause an increase in elimination of lead in the urine in each one of a random sample of nine cases such as are discussed in this paper. It is clear, then, that the taking of sodium citrate was the cause of the improvement in clinical condition and in the results of the laboratory tests mentioned and the increased elimination of lead in the urine.

Summary.

1. Sodium citrate (British Pharmacopœia) in oral doses of four or five grammes taken in an ounce or two of water three times a day is a valuable and simple method of treatment of lead poisoning.

2. It causes a rapid decrease in symptoms and improvement in general clinical condition.

3. It causes increased elimination of lead in the urine without concomitant exacerbation of symptoms.

4. It is simple and easy to take and causes no unpleasant effects, beyond slight occasional nausea; but this effect has been noticed in very few cases.

5. This paper confirms the general conclusion of Kety and Letonoff as to the value of sodium citrate in the treatment of lead poisoning.

Acknowledgements.

Acknowledgements are gratefully rendered to Dr. W. W. S. Johnston and Dr. K. D. Fairley for access to cases in

the Royal Melbourne Hospital; to Dr. A. T. Taylor and Dr. R. Charlton for referring cases to this division and for clinical notes; to the State Laboratory for the analyses for arsenic, and to Dr. A. C. Christophers, B.Sc., for his interest in the work; to Associate Professor Belz, associate professor of mathematical statistics in the University of Melbourne; and to Mr. W. Challinor and Miss L. B. Blair for preparing the graphs.

References.

Ferguson, A. E., and Ferguson, T. (1934), "The Examination of Blood Films in Relation to the Prevention of Plumbism among Ship-breakers", *Bulletin of Hygiene*, Volume XXXIV, page 295.

Fisher, R. A. (1926), "Statistical Methods for Research Workers", Edinburgh.

——— and Yates, F., "Statistical Tables".

Kety, S. S. (1942), "The Lead Citrate Complex Ion and its Role in the Physiology and Therapy of Lead Poisoning", *The Journal of Biological Chemistry*, Volume CXLII, page 181.

——— and Letonoff, T. V. (1943), "Treatment of Lead Poisoning with Sodium Citrate", *The American Journal of the Medical Sciences*, Volume CCV, page 406.

Letonoff, T. V., and Kety, S. S. (1943), "The Effect of Sodium Citrate Administration on the Excretion of Lead in Urine and Fæces", *The Journal of Pharmacology and Experimental Therapeutics*, Volume LXXVII, page 151.

Shiels, D. O. (1936), "The Concentration of Lead in the Urine of Workers at Mount Isa Mines, Queensland, with Special Reference to its Value in the Diagnosis of Lead Poisoning", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 559.

——— (1937), "Comparison of Punctate Basophilia and Ratio of Large and Small Lymphocytes in Diagnosis and Prevention of Lead Poisoning", *ibidem*, Volume I, page 535.

——— (1938), "A Rapid Simple Method for the Determination of Lead in Small Quantities of Urine", *The Journal of Industrial Hygiene and Toxicology*, Volume XX, page 581.

——— (1950), "The Influence of Lead Absorption on Ratio of Large to Small Lymphoid Cells", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 205.

Taylor, H. B. (1925), "Lead Poisoning", extract from report of Director-General of Public Health, New South Wales, Section 1C.

SHOULD WE TREAT LEUCHÆMIA IN CHILDHOOD?[1]

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LEUCHÆMIA at any age is a disease invariably fatal and in its terminal stages often distressing. In children it almost always occurs in the acute form, with consequences that are particularly tragic. There was until recently but one blessing—that the illness at least was generally very brief.

In the past two decades, however, the prognosis for children with acute leuchæmia has been altered by the use of blood transfusions, sulphonamides and penicillin. In 1948 Diamond observed that partial or complete remissions occurred in 10% of 300 cases, the average duration of the remissions being slightly less than ten weeks. Since then other antibiotics have been added to our armamentarium, but even so it has rarely been possible to delay the fatal outcome for more than a few weeks or months. Most doctors, including ourselves, when confronted with a child suffering from acute leuchæmia, still felt that the treatment was worse than the disease and that the alleviation of distress was all that should be attempted.

This was the position in June, 1948, when Sydney Farber, of Boston Children's Hospital, published the results of a year's experience with the use of folic acid antagonists in acute leuchæmia (Farber *et alii*, 1948). Improvement occurred in 10 of 16 children so treated. In another paper in 1949 Farber stated that at least 50% of 60 leuchæmic children showed clinical and hæmatological improvement attributable to the action of these drugs. Two of the surviving patients had been under treatment for long periods, one for twenty-three and one for sixteen months. More recently several other workers have published results which in the main confirm Farber's findings. No claim has been made that these drugs provide a cure, but it is apparent that their effects justify a reconsideration of the question: should we treat leuchæmia in childhood? This is of importance to the physician and to the general practitioner who has the responsibility of a child diagnosed as suffering from leuchæmia.

We consider this question cannot be answered without a thorough knowledge of the natural course of leuchæmia in childhood and of the effects both good and bad of the newer therapeutic weapons. Farber's first paper dealt in detail with only five cases, selected without controls. Even in the later reports by Farber and Dameshek in February, 1949, there was no mention of controls in a combined total of 95 cases studied. It is generally true, moreover, that the initial claims for a new drug tend to be extravagant. To answer this question, therefore, it was decided at the Children's Hospital, Melbourne, late in 1948, to carry out a systematic trial of the effects of the most readily obtainable folic acid antagonist, namely, aminopterin, and that trial forms the basis of this paper. This drug was kindly supplied from Lederle Laboratories, New York, through Dr. John McLean, who was the first to use it in Australia. Other compounds closely related to aminopterin have been tried—amethopterin, aninopterin and amino-an-fol; but in appropriate dosage their therapeutic and toxic effects have proved to be no different from those of aminopterin.

Present Investigation.

In the investigation at the Children's Hospital, Melbourne, a strenuous attempt was made to establish proper controls and to avoid any bias in the selection of cases. The method of randomization was chosen to determine which patients should be treated with aminopterin. Unfortunately it was not possible to adhere strictly to this intended plan throughout the investigation. This was to a slight degree due to uncertain supplies of the drug in the early stages, but it was mainly the result of the high reputation which aminopterin enjoyed at one period, so that the expressed wishes of the parents sometimes made it obligatory to use this drug. It must be stressed, however, that at no time was any selection of patients for aminopterin treatment made on clinical grounds. Moreover, if the parent's wishes produced any bias in the selection of patients, we consider it more likely that this would have favoured the use of the drug in the most serious cases rather than the reverse.

Throughout the investigation all the patients were managed and observed by one person. In each case before treatment records were made of the full clinical findings, the weight, and any evidence of infection, and a full examination of the blood and of the bone marrow was carried out. Progress was checked by records made at least once a week of the blood picture, the weight, the size of the liver, spleen and lymph glands, and the presence of hæmorrhagic phenomena. Bone marrow examinations were repeated every two to four weeks at first, and thereafter as indicated.

The treatment in all cases included blood transfusions whenever the hæmoglobin level fell below 60% to 65% (8·7 to 9·4 grammes per 100 millilitres). Febrile episodes were treated vigorously with penicillin, often with the addition of streptomycin and sometimes of sulphadimidine. Penicillin was also given prophylactically so long as the absolute count of neutrophile cells was below 1200 per cubic millimetre. Complete bed rest was found to be important until the appetite was satisfactory and the weight rising. Every effort was made to give a diet rich in protein and in vitamin *B*.

Aminopterin, when used, was given by intramuscular injection during the first year, and by mouth in recent months when the tablets became available. The drug appeared to produce comparable effects, milligramme for milligramme, whether given orally or parenterally, provided the patient was not vomiting. The most appropriate dosage to use was determined with much difficulty,

[1] Read at a meeting of the Section of Pædiatrics, Australasian Medical Congress (British Medical Association), Seventh Session, Brisbane, May-June, 1950.

[2] Working partly with the assistance of a grant from the Department of Pathology, University of Melbourne.

for reasons outlined below. In the earlier period of trial and error, ignorance led to some unfortunate results. In retrospect it is considered that several patients were not given an adequate amount of aminopterin. On the other hand, the drug is extremely toxic, and it is certain that two of the first eight patients were the victims of a fatal dose a fortnight after commencing treatment, while others narrowly escaped a similar outcome. We have now adopted the following provisional plan of dosage: 1·75 to 2·0 milligrammes per stone of body weight per week for the first week; 1·25 to 1·5 milligrammes per stone per week for the second and third weeks; then 1·0 milligramme per stone per week as a maintenance dose. A close watch was kept for evidence of toxic effects, which calls for an immediate adjustment of the dosage. With the above plan of dosage toxic reactions often occurred and it was necessary to discontinue the drug for one to two weeks. Our experience has provided some support for the view expressed by Dameshek that "to obtain a remission it was necessary to bring about definite so-called toxic manifestations".

Aminopterin, or 4-aminopteroylglutamic acid, is an analogue of folic acid. It was described by Farber as "a biological antagonist to folic acid", but its mode of action is still uncertain. In man it is such a dangerous drug that its sale has not as yet been permitted even in America. Its dangers lie in the facts (*a*) that it is extremely toxic, (*b*) that, like digitalis, it appears to us to be a cumulative drug, although no reference to this has been found in the literature, and (*c*) that all the clinical manifestations of its toxic effects can also be produced by leuchæmia itself, so that early recognition of overdosage may be extraordinarily difficult. In our experience an excessive or toxic dose will not produce serious effects until two or three days later, and a fatal outcome is most likely to occur four to five days after administration of the drug has ceased. Moreover, the margin of safety between the optimal therapeutic dose and the fatal toxic dose is generally very small. We have found the earliest manifestations of a toxic reaction to be anorexia and stomatitis, followed usually by pallor in excess of what would be expected from the patient's hæmoglobin level, and by nausea, vomiting and abdominal pain. A severe toxic reaction always includes an increased tendency to hæmorrhage especially from the intestines, and the hæmatopoietic tissue of the bone marrow is likely to become hypoplastic.

In the present investigation a study has been made of 26 leuchæmic children referred to one of us (J.H.C.) since the middle of 1948. Five of these were private patients, of whom four were treated in the Children's Hospital. The type of leuchæmia was lymphatic in twenty cases, myeloid in three and monocytic in three. Of the 26 patients, 18 were treated with aminopterin and eight were in the control series. As has already been indicated, no selection of patients for treatment with aminopterin was made on clinical grounds; nor was any attempt made to differentiate between the so-called acute, subacute and chronic forms. None of the patients would have been regarded by earlier authorities as having chronic leuchæmia, with the possible exception of one child who, as it happens, did not receive aminopterin. Irradiation of the spleen was used in two relatively resistant cases of the aminopterin-treated series; urethane was used in one case of the control series. These agents did not appear to produce either appreciable symptomatic relief or prolongation of life.

Method of Assessing Results.

The assessment of the results of the treatment of acute leuchæmia may be based on (*a*) improvement of the clinical condition with remission of symptoms, (*b*) improvement of the peripheral blood picture and of the bone marrow findings, and (*c*) duration of life. With regard to the first of these criteria, the occurrence of a clinical remission with regression in the size of the liver, spleen and lymph glands, we have adopted different standards from those recently described by Dameshek (1949). As a general rule, when a remission of symptoms has occurred the patient has not been discharged from hospital until his weight has been rising and he has shown a platelet count above 40,000 per cubic millimetre, a neutrophile cell count above 1200 per cubic millimetre, and signs of ability to maintain a hæmoglobin level above 60% (8·7 grammes per 100 millilitres). On occasions for humanitarian reasons a patient has been discharged from hospital without having attained these levels, and in such cases readmission has almost always been necessary within a matter of days. Arbitrarily, we have regarded a period at home of more than two weeks and less than six weeks as a short remission, and a period of six weeks or more as a long remission.

Improvement in the peripheral blood picture was considered to have occurred if, for two weeks or more, there was an increase in the hæmoglobin value, erythrocyte count, and numbers of neutrophile cells and platelets. Improvement in the bone marrow picture was indicated by a reduction in the total nucleated cell count and in the percentage of leuchæmic cells, and by an appreciable degree of recovery of the myeloid and erythroblastic series. All the bone marrow examinations were made either by one of us (A.L.W.) or by Dr. John Perry, pathologist to the Children's Hospital, Melbourne.

The duration of life or period of survival is regarded by Farber (1949) as "the simplest criterion of therapeutic effect". In our investigation this was computed from the day the patient was first admitted to hospital with the diagnosis of leuchæmia.

Improvement produced by aminopterin is not usually evident until ten to fourteen days after the commencement of treatment. Of our group of 26 patients, six (three treated with aminopterin and three controls) died within the first fourteen days. For the purpose of computing the benefits of aminopterin such cases have been disregarded, as has been the practice of the American workers. Of the remaining 20 patients, five are still alive at the present time,[1] after periods of treatment ranging from two months to eleven months.

Before we submit the results of the present investigation, it may be profitable first to consider what was the usual outcome for leuchæmic children admitted to hospital prior to the advent of folic acid antagonists. In the ten-year period from July, 1938, to June, 1948, 94 patients suffering from leuchæmia were admitted to the Children's Hospital, Melbourne, and of these 65 eventually died in hospital. This number covers all cases of leuchæmia, both acute and chronic, and it includes one child who survived for twenty-two months with chronic reticulosis which was dubiously labelled "atypical monocytic leuchæmia". Table I shows that from the date of the first admission to hospital the average period of survival for these 65 patients was a little over seven weeks, and if the doubtful case just referred to was excluded the average would be five and a half weeks. Five patients (two with lymphatic, two with myeloid and one with monocytic leuchæmia) survived for more than three months—two for nine and a half months, one for six months, one for five months and one for three and a half months. Blood transfusion was of course employed for many of these patients, and some received penicillin as well.

These figures make interesting reading, but for many reasons they cannot be regarded as comparable in any way with those from the 1948-1950 series of patients who received more intensive therapy and detailed attention by one individual. Effective means for controlling the infections that complicate leuchæmia undoubtedly result in a considerably longer period of survival, and such means were certainly not available throughout the ten-year period here described.

Results.

The present investigation is as yet incomplete and the number of cases is small. We believe, however, that it is justifiable to present the findings to date as a preliminary report. The results are shown in Tables II, III and IV.

Clinical remissions occurred in 13 of 15 cases in the aminopterin series and in three of five cases in the control series (Table II). The remissions were long in nearly half the cases in each series. In three of the aminopterin-treated cases there was more than one remission, and in one of the control cases there were several partial remis-

[1] May 5, 1950.

sions. The six patients who died within the first two weeks have been omitted from these tables, for reasons already stated. If they had been included in Table II, the remission rate would have been 72% in the aminopterin series and 37·5% in the control series. These figures probably give a more accurate indication of the effectiveness of aminopterin. A remission occurred in seven of the last eight patients treated with aminopterin, and the eighth patient was symptom-free for a period of a month

TABLE I.

Average Survival Period of Leuchæmic Children who Died in Hospital, 1938 to 1948.[1]

Type of Leuchæmia.	All Patients.	Patients Surviving for Two Weeks.
Lymphatic	6·1 (33)	10·6 (17)
Myeloid	7·0 (10)	8·3 (8)
Monocytic	49·0 (3)	49·0 (3)
Unspecified	2·25 (19)	4·0 (6)
All types	7·2—[5·5][2] (65)	12·3—[9·8][2] (34)

[1] Figures in parentheses indicate the number of patients treated.

[2] These are the figures obtained when one doubtful chronic leuchæmic patient is excluded.

and spent ten days at home, even though the dosage of the drug was suboptimal. In this investigation a clinical remission as defined above enabled the child to enjoy life with his family at home, and in the long remissions most normal activities were possible. One child was allowed to return to school, but this was not usually advised because of the increased risk of infection.

TABLE II.

Clinical Remissions.[1]

Subjects.	Aminopterin Series: Remissions.			Control Series: Remissions.		
	Long.	Short.	Total.	Long.	Short.	Total.
Deceased (15) ..	5	4	9[2] (11)	1	1	2 (4)
Surviving (5) ..	1	3	4 (4)	1	0	1[3] (1)
All subjects ..	6	7	13 (15)	2	1	3 (5)

[1] Figures in parentheses indicate the number of subjects treated.

[2] Three of these patients had two remissions.

[3] This patient had several partial remissions.

Improvement in the peripheral blood picture (Table III) occurred in all 15 cases in the aminopterin series, although in four of these it was only partial and was not progressive for more than three or four weeks, possibly owing to inadequate dosage. In the control series some improvement in the blood picture occurred in all five cases, but in two of these it was not only very limited in degree but also extremely short-lived.

Improvement in the bone marrow picture occurred in all but two of the aminopterin-treated patients for whom serial marrow punctures were performed, and in these two cases the dosage again was apparently too small. In the control series insufficient follow-up marrow punctures have been carried out; but in one case three serial examinations did not reveal any improvement.

The average period of survival in each series is shown in Table IV. In this analysis patients still alive cannot usefully be considered. Their data are mentioned in the table only for the purpose of indicating that one of the control patients has already survived for eleven months without aminopterin. For the deceased patients the average period of survival in the aminopterin series was twenty-one weeks and in the control series 10·4 weeks.

Dameshek (1949) found that the best results with aminopterin were obtained in lymphoblastic leuchæmia, and that has also been our experience. In nine cases of lymphatic leuchæmia the average period of survival was almost twenty-three weeks or five and a quarter months. Survival of three months or longer has occurred so far in thirteen out of a total of eighteen cases in the aminopterin series and in two out of eight cases in the control series.

TABLE III.

Hæmatological Remissions.[1]

Subjects.	Aminopterin Series: Remissions.			Control Series: Remissions.		
	Complete.[2]	Partial.	Total.	Complete.[2]	Partial.	Total.
Deceased (15) ..	6	5	11 (11)	2	2	4 (4)
Surviving (5) ..	3	1	4 (4)	—	1	1 (1)
All cases.. ..	9	6	15 (15)	2	3	5 (5)

[1] Figures in parentheses indicate the number of patients treated.

[2] "Complete remission" means a return of approximately normal figures for red cell count, hæmoglobin value, white cell count, neutrophile cell count, lymphocyte count and platelet count.

Conclusion.

What valid conclusions can be drawn from these observations?

The clinical impression has been that aminopterin definitely causes temporary regression of the leuchæmic process in the great majority of children, as shown by

TABLE IV.

Average Period of Survival in Patients Alive after Two Weeks.[1]

Subjects.	Aminopterin Series: Survival.	Control Series: Survival.
Deceased (15)	21·0 weeks (11)	10·4 weeks (4)
Surviving at May 5, 1950 (5)	14·8 weeks (4)	47·0 weeks (1)
Lymphatic leuchæmia: deceased (13)	22·9 weeks (9)	10·4 weeks (4)

[1] Figures in parentheses indicate the number of patients treated. Survival period was computed from the date of the first admission to hospital.

the remission of symptoms, by regression in size of the liver, spleen and lymph glands, by improvement in the blood and bone marrow pictures, and by prolongation of life beyond the period to be expected from previous experience of this disease. That the drug has not been more effective in some cases may have been due to inadequate dosage, or to relative resistance to its effect on the part of some types of leuchæmia. Monocytic leuchæmia, myeloid leuchæmia and the more chronic forms of lymphatic leuchæmia certainly appear to be less influenced by the drug than is acute lymphatic leuchæmia.

The control series in this investigation has also shown a greater incidence of remissions and a longer average period of survival than was expected from a study of the literature and of the earlier cases at the Children's Hospital, Melbourne. It appears, therefore, that assiduous attention to symptomatic treatment with blood transfusions, anti-infective agents, diet *et cetera* can prolong life considerably, if such is desired. It is also obvious that no reliable assessment of a new therapeutic agent for leuchæmia can be attempted unless appropriate arrangements are made for controlling the investigations.

Some of our results have not yet any statistical value, but the investigation is still incomplete. That aminopterin has a deterrent effect on the multiplication of leuchæmic cells cannot, however, be doubted. This was shown most

dramatically in the case of the two patients who unwittingly were given a fatal overdose and died within two weeks. The story of one of these patients is worth recording.

A boy, aged three and a half years, had acute lymphatic leuchæmia. On his admission to hospital his liver and spleen were enlarged to 5·0 to 6·5 centimetres below the costal margin, and he had generalized glandular enlargement producing in the face a Mikulicz syndrome of considerable degree. Within twelve days of his starting aminopterin treatment, his liver, spleen and glands had returned completely to normal size, the Mikulicz syndrome had vanished, and the leucocyte count had dropped from 140,000 to 1000 per cubic millimetre; but he died on the fourteenth day. At autopsy there were only scanty signs of leuchæmic infiltration, but the bowel was extensively denuded of its mucosa.

In conclusion, we would say that in leuchæmic children the most careful attention to the relief of pancytopenia by blood transfusions, to the prevention and early treatment of infective complications by the best available agents, and to a specific attack on the leuchæmic process by folic acid antagonists will result in a high incidence of remissions and in an average period of survival lasting, at least in lymphatic leuchæmia, for five to six months. Is such an achievement desirable?

It cannot be too strongly emphasized that the folic acid antagonists are exceedingly toxic and their use should be restricted to institutions in which experienced observers and the fullest hæmatological facilities are available. However, there are already glimmers of other and perhaps better therapeutic agents on the horizon. In view of this, we submit that it is best to put the facts to the parents and leave to them the decision regarding treatment.

Summary.

1. The changing outlook for children with leuchæmia is shown to justify a reconsideration of the question: should we treat leuchæmia in childhood?

2. An account is given of an investigation at the Children's Hospital, Melbourne, into the effect of aminopterin in leuchæmia, with a description of the method of assessing results.

3. A preliminary report is given of the findings in 26 cases of leuchæmia—eighteen patients treated with aminopterin and eight controls.

4. Clinical remissions occurred in the great majority of cases in the aminopterin series and in three cases of the control series. Hæmatological improvement occurred in all cases, but was of greater degree and duration in the aminopterin series. The average period of survival in the aminopterin series (twenty-one weeks) was longer than in the control series (10·4 weeks).

5. The decision to treat or not to treat the child suffering from leuchæmia should be made by the parents after they have been fully acquainted with the facts.

Acknowledgements.

We are indebted to the honorary physicians of the Children's Hospital, Melbourne, for permission to study patients under their care and for their whole-hearted cooperation during the investigation. We are very grateful to Dr. John Perry, pathologist to the hospital, for organizing the necessary laboratory facilities in the first place, and to the technicians, Sister Morris and Sister Riley, for their painstaking and conscientious studies of the blood. We also express our appreciation of the efforts of the resident medical officers and ward sisters, who were called upon to do a great deal of additional work.

References.

Dameshek, W. (1949), "The Use of Folic Acid Antagonists in the Treatment of Acute and Subacute Leukemia", *Blood*, Volume IV, page 168.

Farber, S., Diamond, L. K., Mercer, R. D., Sylvester, R. F., and Wolff, J. A. (1948), "Temporary Remissions in Acute Leukemia in Children Produced by Folic Acid Antagonist, 4-Aminopteroyl-glutamic Acid (Aminopterin)", *The New England Journal of Medicine*, Volume CCXXXVIII, page 787.

Farber, S. (1949), "Some Observations on the Effect of Folic Acid Antagonists on Acute Leukemia and Other Forms of Incurable Cancer", *Blood*, Volume IV, page 160.

ON THE MANAGEMENT OF SEVERE BURNS IN THE "TARAKAN" EXPLOSION.

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Introductory Observations.

THE management of severe burns is always a difficult therapeutic problem if death is to be prevented and disfigurement minimized. The organization of an institution and the skill of medical attendants may be taxed considerably. The programme adopted in the treatment of casualties from the *Tarakan* explosion may therefore be of interest to those called upon to treat such subjects. It is worthy of comment that the treatment is within the scope of any hospital.

For the guidance of resident medical officers, an outline of the chief aims was stated at the outset and emphasis was laid upon the following considerations.

1. Prevention of shock. (i) Do not undress severely burned patients in the casualty department—admit them to hospital rapidly. (ii) Administer morphine. (iii) Commence the intravenous administration of serum at once; do not wait for hæmoconcentration. (iv) Commence chemotherapy at once.

2. Dressing of the burns. (i) Do not administer general anæsthetics. (ii) Mask all attendants and the patient. (iii) Dress a section of the burned patient at a time, and only after resuscitation is well established. (iv) Clean the area with detergent and dress it. (v) The best dressings are as follows: (*a*) for flat areas, coagulation with gentian violet and silver nitrate applications; (*b*) for the hands, the Bunyan bag; (*c*) for the face, 2% benzocaine ointment on a sterile lint mask; (*d*) for flexures and limbs, pressure dressings of fluffed sterile wool over 2% benzocaine ointment gauze. (vi) Three to four hours later a further section of the dressing may be carried out.

3. Associated injuries. These must be dealt with in correct perspective and not allowed to interfere with the correct management of the burns.

4. Prevention of dehydration. (i) Continue the intravenous administration of serum until the urinary output it maintained at more than 40 ounces *per diem* (this period may be thirty-six to forty-eight hours), and until the hæmoglobin value or white cell count is normal. (ii) Pay frequent attention to the dressings; good coagula and firm pressure dressings assist in preventing fluid loss.

5. Prognosis. Do not give a prognosis until the critical period (five to seven days) is past.

6. Grafting. Grafting for unhealed burns is an activity of the second and third weeks, and all plans should be made with a view to securing complete healing in four weeks.

Emergency Treatment.

Thirteen casualties were diverted to Sydney Hospital at fifteen minutes' notice from a "between-decks" explosion on *L.S.T. Tarakan* at about 8 a.m. on January 25, 1950. Three wards were immediately notified that they would be required to receive casualties, and further staff was allocated from less busy parts of the hospital. At the same time ancillary units such as the hospital kitchen and laundry were informed. The first casualties arrived at the hospital by means of the Central District Ambulance just

after 8.30 a.m. The patients were received in the hospital casualty department, where trolleys were ready for their immediate transport to the wards.

From the time of arrival in the casualty department until their discharge from hospital, the treatment of the burned casualties can be divided into the following sections: (i) first aid treatment in the casualty department; (ii) immediate treatment of shock and general resuscitation in the ward; (iii) treatment of burned areas; (iv) treatment of associated injuries; (v) prevention of fluid loss and replacement of lost fluid; (vi) restoration of function.

First Aid Treatment in Casualty Department.

On their arrival at the casualty department, all patients were given a hypodermic injection of morphine sulphate (a quarter of a grain), were covered with a sterile sheet and blankets, and immediately dispatched to the ward. As far as possible one of the standard hospital admission sheets was sent to the ward with each patient, setting out the amount of morphine given and the name of the patient, if identification was possible. These patients, all of whom were healthy young males, had already received a quarter of a grain of morphine at the ship's side. In the casualty department all needed an extra quarter of a grain of morphine. No attempt was made in the casualty department to remove clothing or to assess the extent of the burns. The patients were sent alternately to each of the three wards. The use of three wards was found to be beneficial from two main points of view. No one ward received a sudden inrush of burned patients, but each had a reasonable time to deal with each patient as he arrived, the burden of nursing was divided, and the interest of a greater number of the staff was elicited—this being of great importance in maintaining team spirit so necessary in the prolonged treatment of burns.

Immediate Treatment of Shock and General Resuscitation in Ward.

Each of the three wards to which the patients were sent was staffed by a senior resident medical officer, a junior resident medical officer, a sister and the usual number of ward nurses. For the first week after arrival of the burned patients these teams were kept in each of the wards constantly day and night, while at night additional senior nurses were allocated. This was found to be absolutely necessary if adequate treatment was to be carried out. Of the 13 patients received by the hospital, one was dead on arrival in the casualty department, and one died on admission to the ward. The remaining 11 were all conscious on admission to hospital and were showing greater or lesser degrees of shock. Intravenous serum infusion was started immediately and was running satisfactorily in all cases within forty-five minutes of the arrival of the first patient. Each patient, on being put to bed, was covered with a sterile sheet and warm blankets, and the foot of the bed was raised. At this stage no attempt was made to interfere with clothing or with the splints which had been put on by the ambulance officers. Serum was run in at this stage as fast as possible in all cases. Within about an hour and a half the condition of the patients had improved to a degree which permitted a more accurate assessment of the injuries.

Prevention of Fluid Loss and Replacement of Lost Fluid.

When possible, fluid was given by cannula inserted into the long saphenous vein just proximal to the medial malleolus. In some cases burns or associated injury of the legs made it necessary to use veins in the forearm, and in these cases needles were used. Ten patients were so treated, two patients having died on or soon after arrival, and one patient being lightly burned and not requiring such treatment.

Fluid balance charts were kept for all patients, and intravenous therapy was continued until a positive fluid balance had been maintained for not less than forty-eight hours.

The rate of intravenous administration of fluids and the type of fluid administered varied with the following factors. (i) The initial condition of the patient. Those with the more extensive burns or with shock from associated injuries naturally received fluid at a faster rate (140 drops per minute) than the others. (ii) The clinical condition of the patients. This was assessed from time to time either by the honorary medical officer in charge or by a senior member of the resident medical staff. (iii) The figures obtained from serial hæmoglobin estimations. These were made twice a day for the first five days and then daily or as required.

The maximum amount of fluid given intravenously in the first twenty-four hours to any one patient was five litres (R.G.) and in the second twenty-four hours also five litres (P.M.). The maximum amount of fluid given intravenously to any one patient was 13 litres given over a period of seventy-five hours; but a complicating factor in this case was the presence of a severe head injury (fracture of the base of the skull, fractured maxilla and mandible). This patient was unable to take any fluid by mouth after the second twenty-four hours. The minimum amount given to any one surviving patient (burns only) was five litres. The four most severely burned patients respectively received 12, 12, 11·5 and 10 litres of fluid intravenously over a period of approximately sixty hours.

The chief fluid used intravenously was pooled human serum, which comprised 75% of the fluid given intravenously in all cases. Blood was given to nine of the ten patients needing fluid intravenously towards the end of the first twelve hours, the maximum amount given being two litres. It was found that one litre of blood given over a period of six to ten hours arrested the fall in hæmoglobin value in those cases in which signs of hæmodilution were present.

Hæmoconcentration was combated mostly by increasing the rate of serum infusion, and in a few cases 5% glucose solution in N/5 saline solution was used with the idea of maintaining serum chloride concentration. The total amount of fluid given intravenously to ten patients was 95·5 litres given over a period of 100 hours.

Intravenous infusion in all except one of the cases was suspended by the fourth day in hospital, when it was considered that oral intake alone could maintain a positive fluid balance; but this balance had to be policed carefully and a rigid check maintained on fluid intake and output. (The exception was a case of severe burns complicated by a compound fracture of the skull *plus* a Pott's fracture and a fractured *os calcis*.)

Of the three patients who died as a result of their burns and/or injuries, it would appear that in two cases the total amount of fluid administered intravenously was insufficient. J.R., who died twenty-seven hours after his admission to hospital, had received eight litres of fluid intravenously *plus* two and a half litres of fluid orally over that period, the output for the same time being 210 millilitres of urine and 1·5 litres of vomitus. W.H., who died twenty-six hours after his admission to hospital, received six litres of fluid intravenously and four litres orally. His output was 1100 millilitres of urine and 1900 millilitres of vomitus. The amount of fluid given intravenously to these two patients could probably have been increased by at least 50% over the same period. The third patient who died received 13 litres intravenously and 1·5 litres orally in seventy-five hours, his output being 4·5 litres of urine.

The Treatment of the Burned Areas.

No treatment of the burned areas was started before the initial shock had been combated and replacement therapy initiated. Treatment of inaccessible burns was deferred until concomitant fractures were treated.

Débridement and cleansing of burned surfaces was restricted to the opening of large blisters and removal of dead skin and obvious foreign bodies. Saline or "Cetavlon" solution was poured over the burned surfaces, but no scrubbing nor diligent removal of dirt was employed.

The general principle in treating the burned surfaces was to cover this area as quickly and as completely as possible to prevent further fluid loss and air-borne infection.

Two methods were used to cover the burned surfaces: (i) the application of pressure dressings, (ii) coagulation.

The faces, hands, feet, forearms and the legs below the knee were treated with pressure dressings. This treatment consisted of dusting with penicillin powder and the application of a layer of *tulle gras* followed by either paraffin wool or sterile cotton wool firmly bandaged with *crêpe* bandages. Care was taken in the case of the fingers to bandage each finger separately. These dressings were left on as long as was consistent with the patients' comfort, none being removed before the fourth day. The *crêpe* bandages were adjusted as necessary to maintain pressure over the burned areas, and if the bandages appeared to be slipping, a fresh bandage was applied over all without disturbing the dressing in any way.

TABLE I.

Subject.	Injuries.	Fluid Given Intravenously.	Result.
R.G.	Burns, compound fracture of skull, fractured ankle.	Serum 5 litres, glucose-saline solution 5 litres, blood 1 litre.	Recovered.
M.S.	Burns, fracture of skull, compound fracture of tibia and fibula.	Serum 7 litres, glucose-saline solution 4 litres, blood 2 litres.	Died on fourth day.
D.S.	Burns only.	Serum 5 litres.	Recovered.
K.S.	Burns only.	Serum 6 litres, glucose-saline solution 3 litres, blood 1 litre.	Recovered.
F.S.	Burns only.	Serum 8 litres, blood 1 litre.	Recovered.
J.T.R.	Burns only.	Serum 11 litres, blood 1 litre.	Recovered.
P.M.	Burns only.	Serum 9·5 litres, glucose-saline solution 1 litre, blood 1 litre.	Recovered.
J.B.	Burns, fractured calcaneus.	Serum 5 litres, glucose-saline solution 4 litres, blood 1 litre.	Recovered.
W.H.	Burns.	Serum 5 litres, blood 1 litre.	Died 26 hours after admission to hospital.
J.R.	Burns, compound fracture of fibula and tibia.	Serum 7 litres, blood 1 litre.	Died 27 hours after admission to hospital.

Flat surfaces such as chest, back, abdomen, buttocks and thighs were cleansed as above and then sprayed with a 2% aqueous solution of gentian violet, which was then allowed to dry. After this a further spray of 5% silver nitrate solution was applied. Coating with these two sprays was continued until a firm dry coagulum was obtained. It was found at first that this coagulum tended to crack and peel off with movement in bed, and as more fluid exuded from the underlying tissues it was necessary to maintain continued sprayings in most cases about three times a day for the first four days. After this period the coagulum remained firm.

A Bunyan bag was used in one case only, in which there were second degree burns of the fingers and hand, normal saline or one in eight eusol solution being employed as the irrigant for twenty to twenty-two hours and the bag oxygenated for two to four hours each day.

Treatment of Associated Injuries.

Associated injuries of necessity must take second place to the severe burns. No attempt to deal with such injuries was made until all shock and fluid loss had been adequately treated. The first injuries dealt with were the conjunctival abrasions and burns. Most of the casualties suffered from minor burns of the conjunctiva; one or two had some loss of superficial cells. Treatment of the eye injuries was carried out under local anæsthetic, pieces of burned debris were removed and the eye was irrigated with warm saline. "Albucid" or "Butyl-Metaphen" ointment was then applied and also smeared round the eyelids. This treatment was carried on daily until no eye irritation was felt and no corneal staining existed.

In the four cases in which anæsthesia was needed for the treatment of the injuries and burns, the anæsthetic solution used was 0·5% procaine solution made up with 4% dextrose solution in N/5 saline solution. This was given through the transfusion needle already in use, and the solution was run in at a rate of 120 drops per minute until a depression both of respiration and of depth of consciousness was noticed. At this point the rate was slowed to 60 to 90 drops per minute until the end of the dressing. The amount used was approximately 200 millilitres in each case.

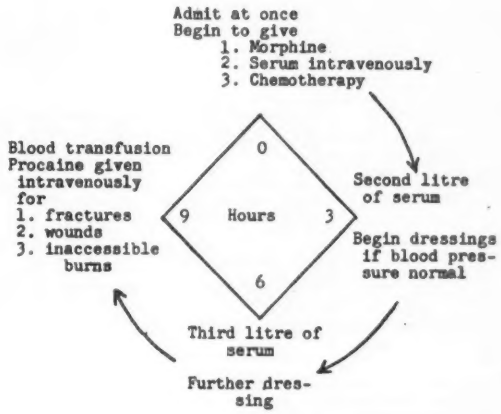

FIGURE I.

The first twelve hours in severe burns.

The only patient of the four who did not have an open injury (R.G., fracture-dislocation of the ankle) needed a greater quantity of anæsthetic solution than the others, by virtue of the fact that the closed injury did not permit of pooling of the anæsthetic, as is the case in open traumatized areas.

This type of anæsthetic proved to be very useful indeed. The patient quickly reached the stage at which compound wounds could be dressed and plaster casts applied. No serious attempt was made to reduce the fractures, it being thought that this could be attempted later when the patient's condition became more satisfactory. The immediate treatment of compound wounds consisted of minimal *débridement*, instillation of penicillin and sulphanilamide powder and loose suturing with either nylon or silk sutures. No *débridement* of skin edges was carried out, as it was thought that this was unnecessary wastage of available skin. Actually both patients with compound fractures died within two days of their admission to hospital, so that the problem of definitive reduction of the fractures in their cases did not arise.

In Case I (J.B.) no treatment for the fractures of the calcaneus and metatarsal head was needed. By the time the burns had healed, both fractures were sufficiently firmly united to allow walking in specially padded shoes. In the case of the calcaneus, all that was needed was excavation of the heel inside the boot and padding with sponge rubber.

In Case XII (R.G.) the problem of the fracture-dislocation of the ankle associated with a depressed fracture of the skull was present. The ankle in the early stages was immobilized in plaster of Paris and at a later date arthrodesis was performed. The depressed fracture of the skull had produced increasing paralysis of the left arm and left

TABLE II.

Case; Patient; Age in Years.	Area Burned.[1]	Burns.			Associated Injuries.	Maximum Hæmoconcentration. (Hæmatocrit).	Result.
		Superficial I.	Superficial II.	Deep.			
I; J.B.; 29	65%	Generalized mixed except where covered by "shorts".		—	Lacerated scalp, fracture of left *os calcis* and of right fourth metatarsal.	44 at 6 hours.	Recovery.
II; P.M.; 26	50%	Face, chest, back.	Arms, hands, conjunctiva.	Arms.	—	48 at 7 hours.	Recovery.
III; J.T.R.; 19	60%		Face, both arms, both hands, upper third of chest; scattered burns over abdomen and back.	—	Internal derangement left knee joint.	46 at 7 hours.	Recovery.
IV; F.S.; 19	50%	Back, left side of chest, left side of abdomen, anterior aspect of legs.	Left side of face, right side of chest, forearms.	—	—	56·5 at 27 hours.	Recovery.
V; K.S.; 21	35%	Face, arms.	Chest.	—	—	43 at 6½ hours.	Recovery.
VI; D.S.; 28	20%	Right side of chest, right hand, right side of face.	Left side of face.	—	—	48 at 7 hours.	Recovery.
VII; P.C.; 45	15%	Face, forearms, hands.	—	—	—	42 at 7 hours.	Recovery.
VIII; L.H.; 39	90%	Both legs, thighs, right arm, neck.	Remainder of body except top of scalp, palms and soles.	—	—	51 at 6 hours.	Died at 27 hours.
IX; F.M.; unknown.	100%	—	—	—	Patient unconscious with fracture of skull and intracranial damage.	—	Dead on arrival.
X; J.R.; 19..	80%	Legs, thighs, arms.	Face, neck, chest, forearms, back.	—	Compound fracture of right tibia and fibula, lacerated right arm.	50 at 9 hours.	Died at 28 hours.
XI; M.S. 39	50%	Abdomen, legs.	Face, chest, arms.	—	Compound fracture of tibia and fibula, maxilla, mandible, and fracture of base of skull with intracranial damage. Patient unconscious throughout.	47 at 6 hours.	Died on fourth day.
XII; R.G.; 23	40%	—	Left side of trunk, left thigh, left arm; right forearm, lower third; right leg; face, neck.	—	Compound depressed fracture of right parietal bone with hemiparesis and third degree fracture-dislocation of right ankle with fracture of *os calcis*.	43 at 7 hours.	Recovery.

[1] Percentage area burned determined by Colebrook's method.

leg. Two schools of thought existed about the treatment of this injury. One was keen to elevate the depressed area immediately, believing not only that early treatment would give a better result as far as the paralysis was concerned, but that the patient's general condition would not improve until it was done. The other school believed that the patient's condition was too poor to attempt any operative procedure, and that the elevation should be left until improvement occurred. It was finally decided to elevate the fracture on January 27, 1950, that is, two and a half days after the patient's admission to hospital. The craniotomy was carried out with intravenous "Novocain" anæsthesia, supplemented by a small amount of "Pentothal" ("Novocain" 1% solution, three grammes, "Pentothal" 0·5 gramme). Pieces of fractured skull were removed from an area about 1·5 inches in diameter. No evidence of extradural or intradural hæmorrhage could be found. The dura was not closed, but the skin was closed completely. A polythene tube was placed through the skin wound down to the opening in the dura for penicillin infusion. The patient made an uninterrupted recovery as far as his craniotomy was concerned, and within forty-eight hours reduction in the paralysis, particularly in the leg, was noted. This paralysis on the day of his discharge from hospital had completely disappeared from his leg, and had diminished in his hand to the extent that he could use it for most purposes, although distinct weaknesses were still present.

Restoration of Function.

Restoration of function proved to be a very important phase of the treatment of the burned patients. One was faced not only with the problem of restoring normal function in injured limbs, but also with the problem of overcoming a strong inclination in many of the patients not to return to the navy. The first problem dealt with was that of restoring function in the burned limbs. This proved to be fairly easy in the majority of cases, owing to the absence of tanning material on the flexures and to the dressing of such burns with "Vaseline" gauze. Penicillin proved of immense help in preventing infection. No problem really arose as far as hands were concerned, except in Case II, in which the patient's left hand had received severe and extensive second degree burns.

As soon as the patient was over the initial stages of shock his hand was placed in the Bunyan bag, which had been packed with finely teased cotton wool. Into this bag saline was run for twenty hours in every twenty-four. Active movement was begun immediately in the bag. For four hours in the twenty-four the limb was allowed to remain out of the bag to dry it. The patient's hand was kept in the Bunyan bag for one week, after which it was removed and dressed with light "Vaseline" gauze dressings. At this stage movement in a paraffin bath was begun. Within about a fortnight the patient's fingers and hand were covered with red, smooth, shiny skin, which rapidly became quite pliable and gave him no trouble in movement.

In all other cases the burns of the hands were not of a very serious nature, but early physiotherapy was instituted and this proved to be successful in preventing any tendency to stiffness.

Comment.

This is a factual report of the treatment afforded to these casualties by the resident and nursing staffs of the Sydney Hospital. A plan for the management of such cases is of value and affords guidance where confusion often reigns.

It is worthy of note that resuscitation precedes treatment. During the phase of hæmoconcentration, serum is, in our view, the infusion of choice; but early blood transfusion is of value to combat hæmodilution.

Local treatment should be designed to avoid shock, fluid loss and infection. It is therefore afforded in divided doses, segments of the burned area being exposed and treated as the patient's condition permits. No local method of treatment is efficient unless it prevents fluid loss.

It is further worthy of note that sepsis did not occur in any of the burns or wounds. This fact is of itself a tribute to the nursing and medical staff who carried out the local treatment.

Penicillin was the sole antibiotic agent employed. Streptomycin was deliberately withheld lest it should be required for the treatment of subsequent infection prior to skin grafting (it was not necessary for this purpose).

The prime objects of treatment—namely, to save life and to have all burns healed in one month—should never be overlooked, and all measures should be devoted to these ends.

The use of procaine analgesia (with or without supplementary "Pentothal") provided a valuable adjunct for the management of associated injuries.

Finally, to the care and enthusiasm of the nursing and resident medical staffs of the wards concerned, many of these patients owe their lives.

BIOCHEMICAL OBSERVATIONS ON CASUALTIES BURNED IN A PETROL EXPLOSION.

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THE main biochemical effects of burns are due to disturbances of water, electrolyte and protein metabolism. Causes of these disturbances are increased loss of water and solutes from the body surfaces, increased protein metabolism, loss of tissue by desquamation, and formation of abnormal products by the direct action of heat on tissues or body fluids, or indirectly by interruption of normal metabolic cycles by heat inactivation of enzymes in the skin (Peters, 1944; Rosenthal and McCarthy, 1947).

Loss occurring from burned surfaces is due chiefly to exudation of fluid containing six to seven grammes of protein per 100 millilitres, and about one-tenth of this concentration of inorganic electrolytes, mainly sodium and chloride ions. As loss of fluid may be at the rate of several litres per day, one obvious result will be dehydration. Losses of protein and of electrolyte, although quantitatively smaller, are not less important. These losses further embarrass fluid balance by disturbance of both colloidal and crystalloidal osmotic pressure, and of ionic balance. In addition loss by insensible perspiration and by sweat from undamaged surfaces may be greatly increased.

The disturbances of fluid and solute metabolism are further aggravated if the loss of circulating fluid is sufficient to impair renal function in the secretion of nitrogenous and other end-products of metabolism in the urine.

Disturbances of protein metabolism other than those due to loss may be due to increase in the rate of metabolism through normal channels (Clark and Peters, 1946). Such increased protein metabolism accompanies other traumatic injuries besides burns (Cuthbertson, 1930). The concentration of urea, the main end-product of protein metabolism, may rise in the blood, and its output in the urine increase. At the same time abnormal metabolites appearing in the blood and the urine increase the residual or undetermined nitrogenous compounds of these fluids. Instead of being a small fraction of the total non-protein nitrogen, they may

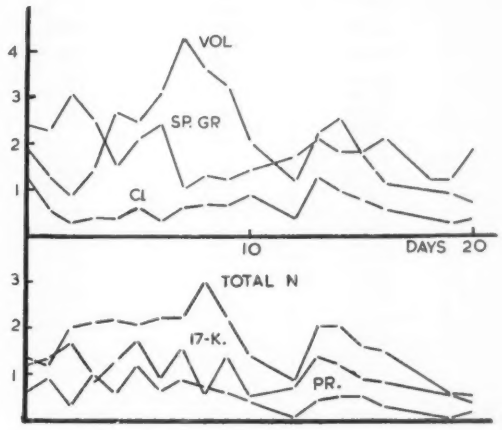

FIGURE I.

Abscissæ: days after admission to hospital. Ordinates, upper graphs: "Vol.", volume of urine per day in litres; "Sp. gr.", specific gravity of urine, 1·0 followed by figures of ordinate; "Cl.", output of chloride in urine per day in grammes. Ordinates, lower graphs: "Total N", output of total nitrogen in urine per day, reading of ordinate × 10 = grammes; "17-K", output of 17-ketosteroids in urine per day, reading of ordinate × 10 = milligrammes; "Pr.", output of protein in urine per day in grammes.

rise to over 80% of it (Taylor *et alii*, 1943). These products form a complex mixture, the composition of which is not yet definitely known. Existing evidence indicates that nitrogenous compounds predominate, whose molecular weight is much lower than that of proteins, but considerably higher than that of normal end-products. The occurrence of numerous imino and amino groups in these abnormal compounds suggests that they may consist largely of polypeptides. They appear to exert their toxic effect by further disorganizing the normal movement of fluid in the tissues.

The increase of nitrogen excreted in the urine is not accompanied by a corresponding increase of potassium (Cope and Nathanson, 1943). This is evidence that the increase of nitrogen is not derived from the breakdown of cellular tissue.

Various indirect effects accompany those mentioned. The more readily observed indirect effects appear to be associated with the action of hormones of the adrenal cortex (Cullumbine and McDonald, 1947). A diminution of carbohydrate tolerance and a raised level of glucose in the blood have been recognized for some time. More recently increased excretion of 17-ketosteroids in the urine has been found. This appears to be associated with increased protein catabolism (Cope and Nathanson, 1943). The converse effect—namely, decrease of protein catabolism on administration of 17-ketosteroids—has been shown by Kochakian and his co-workers (1946). Probably other cortical hormones are involved also. Desoxycorticosterone, for example, has been shown to be concerned in controlling the reabsorption of water from the glomerular filtrate through its effect on the tubular mechanism (Walter and Ingram, 1943).

The present observations were made on eleven casualties burned in an explosion of petrol on H.M.A.S. *Tarakan* on January 25, 1950. The distribution, extent and intensity of the burns and other injuries are stated in the report of Dr. Peter Grant in a paper published in the present issue of this journal. As the observations were made primarily to help in the assessment of the condition of the patients

TABLE I.

Analysis of Twenty-four Hours' Specimen of Urine.

Subject.	Date.	Volume. (Millilitres.)[1]	Specific Gravity.	Chloride. (Grammes.)	Protein. (Grammes.)	Nitrogen. (Grammes.)	17-Ketosteroids. (Milligrammes.)
J.B.	26/1/50	1900 (5000)	1·024	12·8	0·6	13·5	11·9
	27/1/50	1300 (3000)	1·023	5·4	0·9	11·5	13·1
	28/1/50	870 (2000)	1·031	2·8	0·3	20·0	17·1
	29/1/50	1465	1·025	3·8	1·0	21·0	8·0
	30/1/50	2700	1·015	3·6	0·55	21·4	12·8
	31/1/50	2440	1·021	6·2	1·2	20·3	17·0
	1/2/50	3120	1·012	3·0	0·6	21·7	9·0
	2/2/50	4300	1·010	6·0	0·85	22·0	15·5
	3/2/50	3600	1·013	6·9	0·7	30·0	5·0
	4/2/50	3220	1·012	6·4	0·6	21·8	8·8
	5/2/50	2020	1·014	5·3	0·4	13·8	5·0
	7/2/50	1140	1·017	3·5	0	8·2	7·0
	8/2/50	2200	1·021	12·7	0·4	20·3	13·5
	9/2/50	2560	1·018	9·5	0·5	20·4	12·2
	10/2/50	1730	1·018	7·8	0·5	15·7	8·8
	11/2/50	1120	1·021	4·2	0·3	14·8	8·2
	14/2/50	890	1·013	2·4	0	5·5	5·2
	15/2/50	720	1·019	2·3	0·15	5·2	3·8
	20/2/50	2340	1·017	8·6	0·7	12·2	4·3
P.C.	28/1/50	1060	1·007	1·5	0	5·7	—
	29/1/50	2620	1·011	5·0	0·2	6·6	1·7
	30/1/50	1060	1·015	4·1	0·2	5·5	—
	31/1/50 Discharged	1260	1·014	3·9	0·2	7·0	6·6
R.G.	26/1/50	2200 (3500)	1·027	14·6	0·5	14·3	10·7
	27/1/50	930 (4500)	1·022	2·9	0·65	7·9	2·3
	28/1/50	2450 (2000)	1·014	4·7	0·7	22·0	12·0
	29/1/50	2900 (1000)	1·008	3·7	1·15	18·3	24·5
	30/1/50	1470	1·021	3·5	0·4	14·6	7·0
	31/1/50	1100	1·024	2·9	0·9	14·3	—
	3/2/50	3120	1·012	4·2	0·95	22·3	23·0
	4/2/50	1280	1·010	1·4	0·4	12·4	11·8
	5/2/50	2400	1·018	4·2	0·9	22·0	19·7
	7/2/50	2680	1·014	7·3	0·5	17·4	24·0
	8/2/50	1600	1·014	2·7	0·3	15·0	7·8
	9/2/50	3000	1·015	6·9	0·6	20·0	19·5
	10/2/50	1600	1·015	3·6	0·3	12·0	9·6
	11/2/50	1170	1·017	2·1	0·2	11·7	10·3
	14/2/50	1160	1·018	3·1	0·2	14·5	5·6
	15/2/50	1150	1·014	1·5	0·2	6·7	2·8
	20/2/50	2380	1·011	4·2	0·7	16·3	5·2
W.H.	26/1/50	520 (6000)	1·026	2·7	0·4	3·4	6·8
	Died						
P.M.	26/1/50	520 (4500)	1·029	3·9	0·15	4·8	6·8
	27/1/50	— (4500)	—	—	—	—	—
	28/1/50	1545 (2000)	1·017	6·5	0·6	17·8	4·8
	29/1/50	2370	1·012	3·8	5·7	25·2	5·0
	30/1/50	2220	1·022	2·8	5·3	17·8	6·0
	31/1/50	2180	1·016	5·6	3·5	—	3·0
	1/2/50	3360	1·012	6·9	1·7	33·6	8·0
	2/2/50	1170	1·015	3·8	1·75	7·6	3·2
	3/2/50	2100	1·015	2·9	3·4	16·5	4·0
	4/2/50	1020	1·014	1·6	1·6	8·0	4·7
	5/2/50	1720	1·015	2·3	6·8	13·0	8·3
	6/2/50	1200	1·020	1·9	2·4	11·2	3·0
	9/2/50	1160	1·020	4·2	0·3	10·9	3·7
	11/2/50	890	1·024	2·6	0·3	16·6	5·4
	12/2/50	950	1·019	3·3	0·4	7·7	5·4
	13/2/50	920	1·020	3·0	0·3	8·6	3·0
	14/2/50	880	1·021	3·2	0·3	10·5	2·7
	15/2/50	920	1·025	3·9	0·2	10·3	5·0
	17/2/50	840	1·020	3·6	0·2	10·1	4·7
J.R.	26/1/50	116 (8000)	1·019	0·9	0·2	2·2	—
	Died						

[1] Figures in parentheses are the volumes of fluid injected and taken by mouth during periods of intravenous therapy.

and of their response to therapy, they were subject to certain restrictions. Effects observed were the resultant of effects due to burns, to other injuries, and to therapy. Losses of fluid, electrolyte and protein were, for example, balanced in part by intravenous injections of serum. Further, owing to the serious condition of some of the patients, observations were restricted mainly to examinations of urine. Only occasional observations of blood chemistry were possible. Even the regular collection of twenty-four hour specimens of urine at times overtaxed the capabilities of harassed ward staffs.

Observations of the following factors were made on the twenty-four hour specimens of urine received. (i) Daily loss of water in the urine. Some figures are available for the volumes of fluid injected and taken by mouth during periods of intravenous therapy. These figures are shown in parentheses with the corresponding figures for urine in Table I. (ii) Total concentration of solutes in the twenty-four hours specimens of urine as indicated by the specific gravity. (iii) Output of electrolyte as indicated by the output of chloride. Most of the chloride ions in urine are balanced by an equivalent amount of sodium.

TABLE I.—*Continued.*

Analysis of Twenty-four Hours' Specimen of Urine.—Continued.

Subject.	Date.	Volume. (Millilitres.)[1]	Specific Gravity.	Chloride. (Grammes.)	Protein. (Grammes.)	Nitrogen. (Grammes.)	17-Ketosteroids. (Milligrammess.)
J.T.R.	26/1/50	590 (3500)	1·030	3·6	0·8	7·3	—
	27/1/50	1280 (5500)	1·029	5·6	1·8	16·0	16·0
	28/1/50	1700 (2500)	1·022	6·5	3·1	22·8	7·7
	29/1/50	1320 (500)	1·025	3·0	5·3	22·0	19·8
	30/1/50	1450	1·026	1·9	3·5	16·5	13·0
	31/1/50	1800	1·024	2·4	3·3	15·0	17·0
	1/2/50	1720	1·022	1·9	2·9	28·8	5·0
	2/2/50	1210	1·022	1·4	2·2	17·5	8·0
	3/2/50	1380	1·020	1·5	3·5	20·7	13·0
	4/2/50	1180	1·020	2·1	1·9	20·0	5·5
	5/2/50	825	1·020	1·1	0·3	11·5	2·4
	6/2/50	1100	1·024	2·0	0·2	13·9	2·5
	7/2/50	1020	1·025	1·8	0·7	12·7	3·4
	9/2/50	800	1·022	2·9	0·25	8·5	2·6
	10/2/50	1110	1·020	5·3	0·4	12·3	9·5
	12/2/50	1150	1·025	4·4	0·3	11·0	2·7
	14/2/50	720	1·025	3·3	0·2	10·3	5·3
	15/2/50	940	1·025	5·7	0·2	6·7	4·4
	16/2/50	870	1·012	5·8	0·3	10·4	4·1
	20/2/50	1500	1·026	11·4	0·5	16·2	6·8
M.S.	26/1/50	705 (4000)	1·023	1·8	0·65	5·3	8·2
	27/1/50	— (4000)	—	—	—	—	—
	28/1/50	1120 (3500)	1·018	3·5	1·3	16·0	6·6
	Died						
F.S.	26/1/50	610 (3500)	1·025	5·5	0·1	6·9	0·4
	27/1/50	1130 (4000)	1·025	6·2	0·45	12·1	13·0
	28/1/50	1400 (1500)	1·020	6·5	0·4	12·8	12·9
	29/1/50	1950	1·014	2·8	1·0	18·8	10·5
	30/1/50	2860	1·016	4·9	1·1	19·4	5·5
	31/1/50	800	1·014	2·9	0·2	6·2	3·0
	3/2/50	1670	1·020	2·2	0·5	16·0	11·0
	5/2/50	1120	1·013	1·6	0·7	12·0	9·5
	6/2/50	1100	1·018	3·7	0·2	5·3	2·2
	7/2/50	710	1·024	2·5	0·1	5·9	—
	9/2/50	710	1·027	2·3	0·1	10·1	7·4
	10/2/50	840	1·027	4·9	0·2	12·6	1·4
	12/2/50	540	1·020	3·3	0·3	5·4	5·9
K.S.	26/1/50	835 (3500)	1·019	4·2	0·25	5·8	3·9
	27/1/50	— (3500)	—	—	—	—	—
	28/1/50	1700 (3000)	1·013	5·9	0·35	15·4	2·0
	29/1/50	2360	1·009	4·2	0·6	16·0	6·8
	30/1/50	1625	1·014	6·3	0·3	10·6	—
	31/1/50	2150	1·026	5·5	1·1	21·5	14·0
	3/2/50	870	1·020	5·9	0·2	10·3	8·0
D.S.	26/1/50	930 (2500)	1·029	10·3	0·2	[illegible]	5·9
	27/1/50	2545 (1500)	1·015	17·4	0·75	[illegible]	26·5
	28/1/50	1450 (1000)	1·014	8·6	0·4	[illegible]	14·7
	29/1/50	2430	1·011	6·3	1·4	[illegible]	25·7
	30/1/50	1830	1·011	2·3	1·65	10·2	12·9
	31/1/50	2200	1·013	5·2	1·3	16·4	15·0
	3/2/50	2110	1·015	3·2	0·65	16·5	15·0
	4/2/50	610	1·019	2·2	0·2	5·4	9·3
	5/2/50	1200	1·025	3·8	0·7	10·2	19·5
	6/2/50	1420	1·022	7·0	0·4	14·2	11·3

[1] Figures in parentheses are the volumes of fluid injected and taken by mouth during periods of intravenous therapy.

For this reason, and because chloride is so easily estimated, urinary chloride is often assumed to be a measure of the sodium chloride which would be obtained on evaporating urine to dryness. There are, however, objections to this assumption, to which Wynn *et alii* (1950) have recently drawn attention. The potassium-sodium ratio in urine varies about a value of 1:3; this is much higher and more variable than the ratio in blood, 1:20. It must also be borne in mind that salts do not exist as such in solution, but as their constituent ions. Although a given salt may be dissolved in aqueous solution, and recovered from it on evaporation, the statement that the solution "contains" the salt is incorrect. Chloride has therefore been expressed as such and not as the equivalent quantity of sodium chloride. (iv) Protein catabolism as indicated by the total nitrogen content of the urine. (v) Daily output of protein in the urine as an index of possible renal damage. Any such output constitutes a further loss of nitrogen, but the amount is insignificant in this respect. The observations were made by precipitation with salicylsulphonic acid, which precipitates total protein and does not show what proportion is derived from serum albumin. (vi) Output of 17-ketosteroids, the association of which with protein metabolism has been mentioned. (vii) A few scattered observations of the levels of glucose, urea and creatinine. The results obtained were within the normal range of variation.

Observations were continued over a period of twenty-five days. A continuous series of observations was not obtained for each patient for this period for various reasons. Three patients died within three days after their admission to hospital. Apart from the short period for which they were in hospital, their serious condition precluded any measures

not essential for their treatment. Some of these patients had suffered other injuries besides those due to burns. One patient who was not seriously burned was discharged six days after his admission to hospital.

The figures obtained for the composition of the daily urine of the individual patients are shown in Table I.

The figures for J.B., one of the patients showing large daily variations of urinary volume and total nitrogen excretion, have been plotted in the graphs in Figure I.

The information in Table I may be summarized as follows.

Volume of Urine.

In five of the cases investigated the daily volumes of urine rose from the vicinity of 500 millilitres to at least 3000 millilitres. The rise continued for periods ranging from a day to a week. In two cases the initial urinary volumes were about 1000 millilitres per day. These volumes fell to 500 millilitres during the first day after admission to hospital, but rose during the next four to five days to over 4000 millilitres. Very small volumes were received from the patients who died shortly after admission to hospital.

The rapid rise of daily urinary volume was associated with the use of considerable volumes of fluid, usually serum, in intravenous therapy. The high maximal volumes were followed by gradual or fluctuating falls to volumes which became stabilized in the vicinity of 1000 millilitres. These changes were associated with the discontinuance of intravenous therapy.

Specific Gravity of Urine.

The specific gravity of the urine in general followed a course the inverse of that of the daily volume. Initial values in the vicinity of 1·030, associated with the low initial volumes of urine, eventually became stabilized at values in the vicinity of 1·020 when the daily volumes of urine became stabilized at about 1000 millilitres.

Chloride Output.

In general the output of chloride per day followed the variations of the daily volume, so that fluctuations of concentration of chloride were considerably less than fluctuations of output. When there is a deviation from this type of relation it is accompanied by a variation of the daily urinary output of nitrogen in the opposite direction. The resultant of this is that the variation of total solute concentration and of specific gravity is less than that of either of these solutes individually.

Total Nitrogen Output.

Like the output of chloride, the daily output of total nitrogen in the urine varies in the same direction as the volume. The roughly parallel variation of these two chief indicators of solute concentration, derived from electrolyte and nitrogen metabolism respectively, with the volume, suggests that the effect may be one of washing out of solute. This may in turn depend on a diuretic effect of products of nitrogenous metabolites, as well as on variations of fluid intake.

During periods of intravenous therapy these patients received considerable volumes of serum containing over 60 grammes of protein per litre. Injection of five litres of such a fluid in a day would mean an intake of over 300 grammes of protein by this route. This very high rate of protein intake must have its effect on the rate of protein metabolism. It is probably in part responsible for the raised level of nitrogen output in the urine during or following periods of intravenous therapy.

Output of Protein.

Small, and in most cases insignificant, amounts of protein were usually present in the urine. When a rise of total urine nitrogen excretion to more than 20 grammes per day occurred, this was accompanied by a significant rise in the output of total protein. The rise might be to 10 grammes, but was usually not more than five grammes. The protein output fell again when the output of total nitrogen fell below 20 grammes per day. These observations suggest the occurrence of transient glomerular damage during the periods of high total nitrogen output and volume of urine.

Output of 17-Ketosteroids.

The output of 17-ketosteroids in some cases was higher during the first few days after admission to hospital than it was later. Unlike the observations on the victims of the Cocoanut Grove disaster, the increased output of ketosteroids was not observed in every case. The maximal figures did not occur immediately after admission to hospital, but two or three days later. The maxima were not so high, rarely exceeding 20 milligrammes per day as against 25 to 30 milligrammes. The fall to figures below 10 milligrammes per day was not as rapid, but slower and fluctuating.

While there was a general parallelism between daily output of nitrogen and that of 17-ketosteroids in the urine, this was not seen when there were sudden fluctuations of nitrogen output. On these occasions there appeared to be a lag between variation of nitrogen output and that of ketosteroid output.

References.

Clark, E. J., Peters, R. A., and Rossiter, R. J. (1946), "Nitrogen Metabolism after Burning", *Quarterly Journal of Experimental Physiology*, Volume XXXIII, page 113.

Cope, O., Nathanson, I. T., Rourke, G. M., and Wilson, H. (1943), "Management of the Coconut Grove Burns at the Massachusetts General Hospital. Metabolic Observations", *Annals of Surgery*, Volume CXVII, page 937.

Cullumbine, H., McDonald, F., and Simpson, M. M. (1947), "The Role of Leukotoxine in the Production of the Anhydræmia of Burn Shock", *The Journal of Pathology and Bacteriology*, Volume LIX, page 467.

Kochakian, C. D. (1946), "The Protein Anabolic Effects of Steroid Hormones", *Vitamins and Hormones*, Volume IV, page 255.

Peters, R. A. (1944), "The Biochemical Lesion in Chemical Burns", *British Medical Bulletin*, Volume II, page 81.

Rosenthal, O., and McCarthy, M. D. (1947), "Post-Burn Azotemia, Its Characteristics and Relationship to the Severity of Thermal Injury", *The American Journal of Physiology*, Volume CXLVIII, page 365.

Taylor, F. H. L., Levenson, S. M., Davidson, C. S., and Adams, M. (1943), "Abnormal Nitrogen Metabolism in Patients with Thermal Burns", *The New England Journal of Medicine*, Volume CCXXIX, page 853.

Winter, C. A., and Ingram, W. R. (1943), "Observations on the Polyuria Produced by Desoxycorticosterone Acetate", *The American Journal of Physiology*, Volume CXXXIX, page 710.

Wynn, V., Simon, S., Morris, R. J. H., McDonald, I. R., and Denton, D. A. (1950), "The Clinical Significance of Sodium and Potassium Analyses of Biological Fluids: Their Estimation by Flame Spectrophotometry", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 821.

Reviews.

MEDICAL GYNÆCOLOGY.

FEELING that lecturers frequently fail to correlate the academic picture of the lecture room with the symptoms revealed by the patient in the consulting room and that the attempt made to incorporate in clinical teaching important psychological and physiological considerations is insufficient, James C. Janney undertook the task of supplying the need with a book which is meant for medical students and for the practitioner in his consulting room.[1] It is well set up and printed. The illustrations are of high quality, whether reproductions of photographs, diagrams or figures. The contents are divided into seven main parts, each with its appropriate chapters. The sections are: history and physical examination; the patient's complaints; physical findings; tests and special examinations; gynæcological treatment; socio-medical problems in gynæcology; irradiation or operation. The author reminds us that the breasts

[1] "Medical Gynecology", by James C. Janney, M.D., F.A.C.S.; Second Edition; 1950. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9" x 6¼", pp. 462, with 108 illustrations. Price: 61s. 9d.

are gynæcological organs, and that examination thereof should be included in the routine. He recommends transillumination as a means of distinguishing between cystic and solid tumours. As a help in quick reference, most chapters include a précis of the subjects and classification thereof, ruled off in heavy lines. Of particular value to the recent graduate is the advice given in the chapters on dyspareunia, frigidity and the menopause; and in the section devoted to socio-medical problems the author discusses such subjects as preparation for marriage, marital maladjustments, emotional, economic and social factors in marriage, and contraception; there is a chapter devoted to corsets.

This is a somewhat unusual book, but the author concisely covers the ground he had in mind, and it provides a welcome and pleasantly written addition to the general practitioner's library.

A NEW ANATOMICAL ATLAS.

THE production of an atlas of human anatomy is always a major undertaking. Perhaps that is why there are so few atlases at present, for the days when an Albinus could put much of a lifetime and a considerable fortune into such an undertaking are unhappily past. However, a fellow countryman of Albinus—Professor Woerdeman, of Amsterdam—has essayed this task and the first fruits of his labours are embodied in Volume I which has recently been published.[1] The work under review deals with osteology, arthrology and myology. There is no text whatever—the illustrations, which are clear and beautifully reproduced, being left to tell their own story. Such typescript as there is, is in English, while the figures are lettered in Latin. There is a good index. The atlas does not confine itself to the simple task of being an illustration of the human body, for it gives considerable detail in development of bone and particularly of the skull, and here good use has been made of the excellent collection of skulls in Amsterdam to show some of the less common sutural variations. Since there is no text, the atlas can very well be used in conjunction with almost any text-book or course of study, and the student who owns it will find a ready solution to many of his problems. The author and the publishers are to be congratulated on this excellent production.

NEUROPATHOLOGY.

IN their book "Introduction to Neuropathology", the authors, Hicks and Warren,[2] have departed considerably from the usual pattern of such books. Thus one's first impression is perhaps one of disappointment—one would hardly appeal to it as a reference dictionary. When, however, we recognize that the authors had no such end in view, but rather wished to present to the ever-increasing numbers taking courses in psychological medicine a simple, straightforward, coherent and not too long neuropathological accompaniment to their specialty, we heartily endorse their presentation. The paper is excellent and readily reproduces the naked eye appearances and photomicrographs of carefully chosen subjects. Moreover, it is obvious that the authors themselves are practical pathologists and research workers of wide experience, as they find space to illustrate many obscure points long a source of difficulty to fellow pathologists and not often met with in one volume. In clearly demarcated chapters and paragraphs and with useful tables they begin with a short introduction on the pattern of tissue reaction, and this is followed by a concise account of circulatory disturbances and vascular reactions. Inflammatory diseases of wide range are shortly described with references to experimental work. Cerebral malaria, toxoplasmosis and histoplasmosis, meningoencephalitis *et cetera* find a place.

Under the caption "Metabolic, Toxic, and Degenerative Diseases including Insulinism, Alcoholism, Avitaminosis, Demyelinating Disease, Amyotrophies and Myopathies", they present a modern and very necessary account of brain metabolism, dealing with the biochemistry of its sources of energy and its behaviour in deprivations and when body metabolism elsewhere, acquired or hereditary, is abnormal. They closely follow the latest American work on demyelination states and anaphylactic phenomena. They are adherents of the schools which trace the origin of the pia-arachnoid, the Schwann cells and melanophores to the neural crest. On the other hand their treatment of nervous system tumours is most full; they seem to meet with more astrocytomata than is usual, but warn that several pieces of tumour must be examined to exclude glioblastomata. They also find that at times oligogliomata are associated with astrocytomata and ependimomata and would call the small lymphocyte-like cells in pineal, glioblastomata and other tumours neuroblasts. By stringing together a series of tumours of the meningioma, nerve sheath and nævus groups of like cellularity they hold that one may readily trace a suggestive connexion. A useful chapter on developmental errors comes last; the authors hold that perhaps some 20% of products of human conception are developmentally defective, give good illustrations of this, but carefully point out that all tubal embryos are defective. Many other defects are acquired.

RHEUMATIC DISEASE.

IN their book entitled "Manual of the Rheumatic Diseases", W. P. Holbrook and D. F. Hill[1] have succeeded in producing a small concise volume which contains many clinical and therapeutic data, of inestimable importance to the general practitioner, to whom it is dedicated. Their statement that "there is no known single cause and no single specific cure in Rheumatoid Arthritis" is timely and opens the way for the consideration of their full basic programme of treatment of this systemic disease.

As to therapy they still employ gold in rheumatoid arthritis in small doses and maintain its administration for long periods. Almost one-third of the book details the great therapeutic aim, the prevention and correction of deformity. These pages are valuable, justify careful reading and warrant discipleship, as the measures employed by the authors are the result of their experience and success. In dealing with rheumatoid spondylitis the authors stress the necessity of X-ray therapy. They regard cortisone and "A.C.T.H." as great research tools and record the value of small dosage in a series of cases.

The chapter on collagen diseases is timely and important, whilst the benefit of "Benadryl" is of therapeutic significance if corroborated. Short chapters on fibrosis, gout and osteoarthritis follow the usual presentation. The burden of the book is "while we search for a cause and cure prevent deformity".

MODERN TRENDS IN ORTHOPÆDICS.

"MODERN TRENDS IN ORTHOPÆDICS", edited by Sir Harry Platt, covers a wide range of subjects.[2]

The book is introduced by a chapter by the author on "The Evolution and Scope of Modern Orthopædics". Other subjects dealt with are fracture treatment, tuberculosis of bones and joints, pyogenic affections of joints and bones, congenital dislocation of the hip, congenital deformities of the spine and limbs, chronic arthritis, skeleton, joint and muscle tumours, scoliosis, paralysis, injuries and derangements of the spinal column, injuries of muscles and tendons, certain vascular lesions, and bone dystrophies.

Each of these subjects is dealt with by a separate author, each of whom is eminent and experienced in his own subject.

Sir Harry Platt points out that orthopædics today has become one of the two main divisions of general surgery, and represents a large part of the practice of the nineteenth century surgeon. He quotes from Sir Robert Jones, the definition of orthopædics as ". . . the treatment by manipula-

[1] "Atlas of Human Anatomy: Descriptive and Regional", by M. W. Woerdeman, M.D., F.R.N.A.Sc.; Volume I; 1949. London and Australia: Butterworth and Company (Publishers), Limited. 9¼" × 7", pp. approximately 500, with 512 illustrations, some of them coloured. Price: 82s.

[2] "Introduction to Neuropathology", by Samuel Pendleton Hicks, M.D., and Shields Warren, M.D.; 1950. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 9¼" × 7", pp. 508, with many illustrations. Price: $10.00.

[1] "Manual of Rheumatic Diseases", by W. Paul Holbrook, M.D., and Donald F. Hill, M.D., with the assistance of Charles A. L. Stephens, junior, M.D.; 1950. Chicago: The Year Book Publishers, Incorporated. 8" × 5¼", pp. 182, with 119 illustrations. Price: $4.25.

[2] "Modern Trends in Orthopædics", edited by Sir Harry Platt, M.D., M.S., F.R.C.S.; 1950. London and Australia: Butterworth and Company (Publishers), Limited. 9¼" × 7", pp. 504, with 222 illustrations. Price: 59s.

tion, operation, re-education, and rehabilitation, of the injuries and diseases of the locomotor system".

All the articles in the book are well written and the illustrations are plain but good. The standard of the various chapters is so high that it is difficult to make selections on merit. However, for sheer detail, combined with wisdom and ripe experience, the chapter on "Tuberculosis of Bones and Joints", by G. R. Girdlestone, is one which no orthopædic surgeon should fail to read.

It is refreshing to read articles in which the most up-to-date treatment and even treatment which is still on its trial are described in considerable detail and results are compared with older methods.

In the article on "Certain Vascular Lesions", by D. L. Griffiths, the author states that "The orthopædic surgeon may legitimately claim the whole field of vascular disorders of the limbs as part of his territory". This statement will probably be disputed by many general surgeons.

This book should find a place in the library of every orthopædic surgeon, and all other general surgeons who are interested in orthopædics.

ALLERGY.

FROM the use of the word "primer" in the title of the third edition of the work "Primer of Allergy", by Warren T. Vaughan, revised after the author's death by J. Harvey Black, readers might be led to think that the book is more for students than for practising doctors.[1] To some extent, however, the title is a misnomer, for in this work all doctors, whether they are allergists or general practitioners, will find a complete account of all the baffling problems associated with allergy, together with directions for their elucidation.

The book is written in a breezy style and is illustrated by many cartoons. As it is meant to be read by intelligent patients as well as by their physicians, there is little about theory or speculation, the whole tone of the book being essentially practical. All sufferers will find many practical hints in the chapter headed "Directions to the Patient"; some forty-seven pages are devoted to such headings as "Directions for a Person with Allergic Headache", "Directions for Those Sensitive to Cosmetics", and "The Avoidance of Certain Foods", to mention only three of the practical sets of instructions which are fully set out. This book is a valuable contribution to the treatment of allergy and can be confidently recommended.

OLD AGE.

AFTER an interval of six years Trevor Howell has produced a second edition of his book "Old Age".[2] In the compass of eighteen short chapters he has incorporated the result of much original work carried out mainly at the Royal Hospital, Chelsea, and Saint John's Hospital, Battersea. Some of the work has already been published in the *British Medical Journal*, *The Lancet* and journals devoted to geriatrics.

About half the book is devoted to the discussion of the influence of age on the temperature, pulse, hæmoglobin value, arterial thickening, the central nervous system and the kidney. In these chapters there is a review of the work of previous authors together with the result of the author's own observations. It was found that the hæmoglobin level in old age was much higher than it was thought to be by earlier authors. It is a pity that in the discussion of hæmoglobin levels different standards were used for different sections of the investigations. One would have preferred to find all figures expressed in grammes *per centum* rather than to have percentages of varying standards. Good use is made of diagrams and tables to make the text readily comprehensible.

In dealing with rheumatic diseases the author discusses the problem of fibrositis and stresses how frequently patients are wrongly diagnosed as being arthritic. He has found that methods of treatment such as the injection of local anæsthetic agents which are applicable in young people are not so effective in the aged, unless combined with local heat application and massage. He praises the use of such substances as adrenaline, ephedrine and belladonna incorporated in a vanishing cream base for local inunction.

In rheumatoid arthritis the author stresses the high incidence of residual deformities and discusses the various methods of salvage work aimed chiefly at improving the functional status of the patient. His experience with intra-articular injections of procaine lactic acid are interesting. In these he appears to have had considerably greater success than some observers would expect.

Throughout the book sound practical advice is given on the management of the various types of illness met with in old age. The stressing of the importance of an active interest in the problems associated with aging is timely, in view of the growing numbers of elderly people in our community.

Altogether this book is easily read and will well repay careful perusal by all medical men, whether general practitioners or specialists.

A SHORT HISTORY OF PHYSIOLOGY.

IT seems unaccountable that sixteen years should have been allowed to elapse between the publication of the first edition of "A Short History of Physiology" and the appearance of the second edition in 1949.[1] In his book Dr. Kenneth J. Franklin, Professor of Physiology at the Medical College of Saint Bartholomew's Hospital, London, has managed to compress within the space of one hundred and forty pages an orderly array of interesting facts relating to most of the important discoveries in this science from the days of the Greek philosophers to the end of the last century. In the new edition fresh material has been included in the text, which is now interspersed with a series of familiar portraits of pioneer physiologists, and the general format has been altered for the better. All students of medicine should find this small book acceptable for entertainment and scientific enlightenment and handy for easy reference.

The opening chapters touch lightly on a few of the more rational concepts of the medical philosophers of the ancient world from Alcmaeon of Crotona to Claudius Galen and the abiding influences of the great Alexandrian school. There follows a most interesting chapter in which the author neatly summarizes the strangely accurate theories relating to the supposed course of the pulmonary circulation advanced by certain unorthodox individuals up to the time of William Harvey. Since Sir William Osler stressed the point in one of his delightful essays it has been generally accepted that Michael Servetus was the first to give some indication that Galen's ideas on the subject were inconsistent with the anatomical and physiological facts. In recent years, however, an Egyptian physician has been able to produce evidence, subsequently confirmed by Max Meyerhof in 1935, that an eminent Arabian physician, who was chief of the great Nasiri Hospital in Cairo during the thirteenth century, gave an almost modern interpretation of the lesser circulation in his "Commentary on the Canon of Avicenna", nearly three centuries before the *"Christianismi Restitutio"* of Servetus came under the critical notice of John Calvin during the Reformation. And curiously enough, neither Ibn an-Nafis nor Servetus could have arrived at his conclusions by way of the experimental method.

The succeeding chapters direct attention to the vast importance of the work of the anatomists of the Renaissance period in laying the foundation for a better understanding of function once the human and animal bodily structures had been described as they actually occurred in nature. But by far the greater portion of the book is given to the long sequence of discoveries which followed at an increasing rate of acceleration when the experimental method in physiological research came to be recognized as the only way in which natural phenomena could be properly investigated.

The compilation of a wealth of material contained within this small book obviously represents many years of painstaking research, and it is backed by a sound knowledge of the science of physiology.

[1] "Primer of Allergy: A Guide for those who must find their way through the Mazes of this Strange and Tantalizing State", by Warren T. Vaughan, M.S., M.D., revised by J. Harvey Black, M.D.; Third Edition; 1950. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 7½" × 5", pp. 178, with a few illustrations. Price: 36s. 9d.

[2] "Old Age: Some Practical Points in Geriatrics and Gerontology", by Trevor H. Howell, M.R.C.P. (Edinburgh); Second Edition; 1950. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 116, with nine illustrations. Price: 10s. 6d.

[1] "A Short History of Physiology", by Kenneth J. Franklin, D.M., F.R.C.P.; Second Edition; 1949. London: Staples Press, Limited. New York: Staples Press, Incorporated. 8½" × 5½", pp. 154, with a few illustrations. Price: 10s. 6d.

The Medical Journal of Australia

SATURDAY, DECEMBER 16, 1950.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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THE ETHICAL BASIS OF MEDICAL PRACTICE.

A COUPLE of years ago, J. H. Means, Jackson Professor of Clinical Medicine in the Harvard Medical School and chief of the medical services of the Massachusetts General Hospital, wrote to Willard S. Sperry, Dean of the Harvard Divinity School, telling him that on his rounds in the hospital he and his house officers continually came upon problems which belonged in the realm of moral philosophy rather than of medicine. He cited one or two instances and invited Dean Sperry to come and talk to them on the matter. The Dean accepted the invitation and addressed many of the senior members of the staff and most of its junior members. The editor of *The New England Journal of Medicine* asked for the text of the address and published it in December, 1948. The result was a flood of letters to the author, with requests for addresses to the faculty and students in the medical school of the University of Michigan and suggestions that the address should be expanded and published in book form. The book has now appeared.[1] It should be stated at once that this book is written on such a high plane, it is so pregnant with wisdom and earnestness, and withal so easily understood, that it should be brought to the notice of undergraduate medical students by all lecturers in medical ethics.

In his lectures to medical students in the University of Sydney, J. G. Hunter states that the rules and principles concerning moral obligation which are intended to regulate medical practice, constitute medical ethics. This is a suitable definition for students. For non-medical members of the community a distinction has to be made between medical ethics and medical etiquette. Etiquette deals with manners, chiefly in intraprofessional relations. In actual practice manners are inseparable from the moral considerations on which professional conduct is based. Saundby states that it is not easier to find a solid basis for medical ethics than for general ethics; he adds that no undisputed foundation other than the better kind of current opinion has been found. He quotes three principles as the corner-stones of medical ethics. In the practitioner's relation to his colleagues there is the golden rule: "Whatsoever ye would that men should do to you, do ye even so to them." In the practitioner's relation to his patients, their interests should be his highest consideration: "*Aegroti salus suprema lex.*" In his relation to the State, the laws of the country and his civic duties, he has to obey the injunction: "Render, therefore, to Cæsar the things that be Cæsar's." Clearly there is more to medical ethics than obedience to certain rules—the moral considerations which may or may not find expression in set rules must be the determining factor in conduct. This sounds quite obvious and the statement will probably receive general consent. But the spirit of the times is changing and men seek to measure their service as a butcher measures his meat, a barmaid her beer and a draper his yard of calico. There comes to mind a house surgeon at the bedside of a patient on whom he has operated and whose bleeding has not been stopped; the end of his duty period has come and he sends for the senior officer that he may keep an appointment elsewhere. "I am off duty", he says. So he is. He has given the service that butcher, barmaid or draper would give, but he has lost sight of, perhaps he has never appreciated, the moral obligation inherent in the practice of medicine. It is no wonder that Saundby refers to the "better kind" of current opinion. This is what Sperry seeks to drive home in his teaching.

The first chapter in Sperry's book is called "Our Overlapping Professions". That the profession of medicine and that of the ministry of religion overlap is recognized much more today than it used to be. Sperry thinks that the advent of psychosomatic medicine is significant and marks a "concession" on the part of doctors. There is no doubt that the ministry of religion, "wisely used", as Sperry remarks, can do much to temper the suffering of the patient, fortify his will to live and help him if he is to die. On the parson's side, Sperry sees a growing appreciation of the part which bodily processes play in mental health and so in character and conduct. Two of his sentences will bear reproduction: "It used to be said that Carlyle's organ tones of misery were due to duodenal ulcers. Today, Carlyle's melancholy would not be attributed to his ulcers; his ulcers would be attributed to his unresolved emotional relation to Jane Carlyle, his wife." Be that as it may, it is interesting to note that in Sperry's experience theological students of today often sign on for a session at hospital, instead of taking a parish up country or at the seaside. In this session the theological student may work as an orderly and he goes the round of the wards with the chaplain and the house officer. He also sits in at staff meetings "in which doctors, surgeons and chaplains discuss the total problem of the care of the sick, studying individual instances by means of the case method". Sperry holds that in this way the young cleric gains an insight into the mood and manners of the profession of medicine which is of incalculable value to him thereafter. With this most members of the

[1] "The Ethical Basis of Medical Practice", by Willard L. Sperry, with a foreword by J. Howard Means, M.D.; 1950. London and New York: Paul B. Hoeber, Incorporated. 8" x 5¼", pp. 186. Price: $2.50.

medical profession will agree. If they do, they should consent to the corollary—that their treatment of a patient will be enhanced if they have an understanding of the principles and tenets, "the moods and manners", of the patient's religion.

Sperry points out that the medical practitioner is often confronted with problems which are primarily moral, but that he should not approach the situation from the moral aspect unless he is invited to do so. The medical practitioner's profession "presupposes the scientific premise of dispassionateness"; at the same time there is a suspicion that dispassionateness, like patriotism, is not enough. Humanity is endowed with two kinds of mind; one is concerned with the outer world of fact and the other creates a world nearer to its heart's desire than any factual world already to hand. The humanist always finds himself at a disadvantage when he compares himself with the scientist. "The difficulty is that, whereas in science its truths may be verified by repeated experiment, the experiences of life with which the humanist deals can never be repeated." Louis Pasteur once said before the Academy of Medicine in Paris[1] that there were in each of us two individuals, one the man of science who had thrown aside old notions and who by observations, experiments and reasoning desired to obtain a knowledge of Nature, the other the man of feeling, of tradition, faith or doubt who said that the force within him would be transformed. He held that the two domains were distinct and that it was woe to him who in the imperfect state of our knowledge wished to make them encroach one on the other. Sperry admits that the kind of "stubborn schizophrenia" in the modern mind puzzles him. He applies these ideas to the subject of specialization. "Patently we cannot call a halt in specialization, but patently also our highly sophisticated forms of learning cannot be easily equated in terms of an ultimate wisdom for living." The specialist in medicine condemns himself to walk in a narrow sphere or groove; it is difficult for him to avoid monotony and to browse in other spheres. Sperry quotes the epitaph in a little Scotch burying ground which reads: "Here lies the body of Tammas Jones, who was born a man and died a grocer." In the modern world the vocation often swallows up the man. In medicine the pressures are so great upon a man's time and strength that he runs the risk, unless he fights against it, of writing his own epitaph: "Here lies the body of Hiram Smyth, who was born a man and died a gastroenterologist." The importance of these considerations to the question of medical ethics is reflected in a statement by Vannevar Bush (quoted by Sperry) in which are the words: "The doctor, above all men, needs to be a full man." From this Sperry naturally turns to consider the nature of conscience. He quotes a definition of Overholser and Richmond that: "Conscience is not something that is born in the individual, but something that is acquired from the practices, the beliefs, the attitudes and customs of the group with which the individual identifies himself. For the average individual his conscience is the crystallized attitude of his group." Sperry admits that there is a large measure of truth in this sociological account of the origin of conscience. He thinks that this sociological account of conscience, so often invoked by doctors who seek for their patients a well-adjusted life, is at the best a half-truth—it may serve as a point of departure, but it does not exhaust the subject. He discusses the distinction sometimes attempted between ethics and morals. It has been suggested that one of these words should be used to describe the accepted usage of society, and that the other should be reserved for those acts and aims which depart from that usage in the sense that they attempt to go beyond it. Apparently there is no agreement as to which word should be used in which connexion. This very ambiguity suggests, Sperry avers, that the meaning of the word conscience cannot be exhausted by the idea of conformity to the existing social pattern. "There is in the sense of *oughtness* which we associate with the promptings of conscience a plus content which is often well in advance of existing manners." It was this sense of "oughtness" which was lacking in the house surgeon previously mentioned. After stating that the capacity for distinguishing between right and wrong and the necessity for so doing are data commonly accepted, though their concrete applications are variable, Sperry draws an intriguing analogy in the physiological invariability of the life principle in contrast to the prodigal variety of the individuals which it begets and bears. "Hence one is warranted in making a distinction between a wholesome respect for the principle of conscience while accepting the fact that its concrete pronouncements plainly concede the fact of relativity." Those who confess to religious beliefs cannot dissociate morality from religion. Sperry quotes some significant passages from the writings of Father George Tyrell, whom he describes as a "brilliant Catholic Modernist", to the effect that it is faith in God which justifies or explains the absolute values revealed in conscience. Sperry adds: "The exercise of conscience is one of those experiences in which we march along the borders of the ultimate mystery and meaning of human life, where our life touches the being of God." The possession of conscience is peculiarly a human attribute. Additional light may be thrown on the subject by recalling the dictum of a deceased philosopher who was also an Anglican priest to the effect that Jesus Christ, being sinless, had no conscience. The practical point which emerges from this short discussion is that conscience is educable—it may become more acute, more alert; on the other hand it may be smothered and trodden underfoot. We may conclude that one of the main functions of a study of medical ethics is to keep the conscience alert and sensitive.

This brings us to the last aspect of Dean Sperry's book which we propose to mention—the "tragic moral choices" which doctors sometimes have to make. Often, our author holds, the choice is not between absolute right and wrong, between white and black, but between varying shades of grey. (This is what he means by the "fact of relativity".) The question of divided loyalties arises. Instances given by Sperry need not be quoted, but the loyalty to be followed is always that of the welfare of the particular patient and the tradition of devotion to the healing art of medicine—never the welfare of anyone else or, directly or indirectly, of the doctor himself. The old question of motive arises and none but the activator of the motive can know its true nature. Everything else mentioned by Sperry can be discussed under the headings of conscience and motive. We leave this book to the wise discrimination of the medical profession in this country.

[1] Quoted by A. H. Tebbutt, THE MEDICAL JOURNAL OF AUSTRALIA, November 12, 1927, page 671.

Current Comment.

THE USE OF AUREOMYCIN IN HEPATIC DISEASE.

THE wide range of diseases in which favourable results have been obtained from aureomycin has encouraged investigation over a still wider field. One of its interesting features is that it controls not only Gram-positive infections, but certain Gram-negative infections also, and some of the diseases due to viruses. It has been found to exert beneficial control of abdominal infection when given before intraperitoneal operations, and has in addition the valuable property of being effective when given by mouth. It is natural that it should be tried in acute viral hepatitis, whose characters and sequels have been productive of so much useful work during recent years. A research was undertaken by J. M. Shaffer, J. D. Farquhar, J. Stokes and V. M. Sborov in Philadelphia to ascertain if the drug had any curative action in acute viral hepatitis.[1] Alternate epidemic infections were treated with aureomycin, the others being used as controls. There were certain difficulties, in particular the uncertainty of making an early diagnosis, and the notorious variation in severity of the symptoms. Thirty-seven patients were treated for seven to ten days, but comparison with 37 controls did not reveal any significant difference in the average duration of jaundice and bilirubinuria, in the results of flocculation tests or in the clinical picture during the acute and convalescent periods. There were, however, some indications that the late manifestations of the disease were controlled by the drug, and this seemed to warrant further experiments. The hypothesis on which this later work was based depended on the premise that when the liver is damaged by malnutrition or infection, as by a virus, it may be more susceptible to toxic products of viral or bacterial metabolism, or other noxious substances formed as the result of bacterial action on the contents of the intestine. If any support could be found for this supposition, there might be a reason for the use of aureomycin, namely, its power to control certain flora of the intestine.

Accordingly, the scope of the inquiry was widened; some work was also done in Los Angeles, and in addition to the original team, P. György, W. H. Smith, H. Goldblatt, L. W. Bluele, J. R. Neefe, C. M. Whitlock, and J. M. Gambescia assisted in carrying out investigations on the sequels of hepatitis.[2] The first of these studies was made on experimental animals. It was based on the regularity with which hepatic necrosis can be produced in rats by a ration with a low casein content and with no other source of protein than yeast, and on the power of sulphur-containing amino-acids, such as cysteine and methionine, and vitamin *E* to prevent liver damage. It has been suggested that these protective substances may act, not by supplying a deficient food factor, but by a detoxifying action. It may be noted also that the left half of the liver is the seat of much more extensive necrotic changes in animals dying from nutritional liver necrosis than the right. This was attributed by Himsworth to the fact that the right half of the liver receives its portal supply mainly from the small intestine, whereas the left half is supplied by portal veins from the stomach, spleen and large intestine. The protective substances are absorbed from the small intestine, but it is postulated that toxic substances are absorbed from the colon. When rats were fed on a necrogenic yeast diet, aureomycin was found to exert a protective effect which was statistically valid in this series. The number of rats surviving the hepatotoxic agents was greater when aureomycin was given, and further, the onset of liver necrosis was delayed in other animals, even though they eventually died. The surviving rats were found at autopsy later to have normal livers. In some instances scars were seen in the livers of animals which succumbed, suggesting that they recovered from early attacks, but failed to sustain recurrent attacks of hepatitis. There were several questions raised by this result. Was the effect of aureomycin a directly chemical one, or was it antibiotic in action, suppressing infection in the liver, or in the lower bowel? Circumstantial evidence leads the authors to favour the last hypothesis, and in the further work carried out this assumption was made.

The next series of trials was made on patients suffering from acute liver failure, due in two instances to viral hepatitis, and in two to portal cirrhosis. Only four patients were studied, but the findings were encouraging. Full case histories are given in one of the articles. In two cases coma due to acute viral hepatitis occurred; one of these patients was also suffering from myelogenous leuchæmia, and in each instance striking improvement was noted after aureomycin therapy was begun. Both patients recovered from the hepatic toxic state, and remained free from further similar trouble. The other two patients were suffering from portal cirrhosis; one of these, though he succumbed to the disease, was observed to rally from the serious disturbances which he had before after aureomycin was given. This happened several times, and the authors suggest that the phenomenon was too striking to be coincidental. In the concluding study thirteen patients were observed. Nine of them had a clear history of viral hepatitis; the others had chronic hepatic failure due to various causes. The patients had been under observation for some months, and were fairly stabilized in condition. Needle biopsy was carried out in each instance, and a full series of liver function tests was performed before treatment with aureomycin was started, and the usual routine treatment was pursued both before and after the period of trial with the antibiotic. In six patients no effect was observed from the aureomycin, but in the remaining seven, all except one suffering from a chronic sequel of viral hepatitis, definite improvement was noted, the details of which are presented in the case histories. The improvement observed included clinical amelioration, decrease in the size of the liver, and a better response to the liver efficiency tests. One feature of interest was observed in one patient, who manifested sensitivity of the skin, particularly of the hands and nails; as the lesions appeared after exposure to sunlight, it is suggested that aureomycin may sometimes act as a photosensitizing agent.

Some of the evidence presented in these series of trials of aureomycin in chronic hepatic disease may be illusory, and further experience is desirable; but, although the particular use of this antibiotic is different from that originally planned in this research, it seems to be worth repeating.

[1] *The American Journal of the Medical Sciences*, July, 1950.
[2] *Ibidem*, July and August, 1950.

THE SIR RICHARD STAWELL ORATIONS.

SINCE its foundation in 1934, the Sir Richard Stawell Oration has been delivered on sixteen occasions by distinguished men. Founded by Dr. A. E. Rowden White, it commemorates a famous Melbourne physician and clinical teacher, whose influence for good was great, not only in Melbourne but throughout Australia. Recently the founder has had a limited number of copies of the sixteen orations bound and presented to representative Australian libraries. It is proposed that in future the orations will be bound in volume form at ten-yearly intervals. The cost of the volumes will be borne by the founder or his executors, and every ten years a volume will be sent to each of the libraries of the Branches of the British Medical Association at Melbourne, Sydney, Brisbane and Adelaide, to the libraries of The Royal Australasian College of Physicians (Sydney) and Royal Australasian College of Surgeons (Melbourne), to the Rowden White Library, Union House, University of Melbourne, and to THE MEDICAL JOURNAL OF AUSTRALIA. Thus not only will the memory of Sir Richard Stawell be preserved in these libraries, but also, as is stated in the preface to Volume I, the volumes should prove of interest and value to research workers who wish to know the ideas and views of medical scientists for any particular period.

Abstracts from Medical Literature.

THERAPEUTICS.

Rheumatoid Arthritis.

C. M. GUEST, W. H. KAMMERER, R. L. CECIL AND S. A. BERSON (*The Journal of the American Medical Association*, May 27, 1950) discuss the use of epinephrine, pregnenolene and testosterone in the treatment of rheumatoid arthritis. Epinephrine given subcutaneously in doses of 0·5 milligramme four times a day for seven to sixty-five days to 12 patients produced no consistent eosinopenia (such as occurs with cortisone) and produced no beneficial results. Intramuscular injection of pregnenolene daily or two or three times a week resulted in no improvement. Testosterone propionate, 100 to 200 milligrammes given daily or two or three times a week to 71 patients with rheumatoid arthritis, produced no improvement. These investigations were undertaken to test the relationship of glands other than the adrenal cortex to rheumatoid arthritis.

The Common Cold.

D. W. COWAN AND H. S. DIEHL (*The Journal of the American Medical Association*, June, 1950) report investigations into claims that ascorbic acid and antihistaminic agents cure or relieve the common cold. They have dissected the claims of various authors and found little to substantiate them. In their own investigations five groups, each of about 70 students, were treated with ascorbic acid, phenindamine tartrate ("Thephorin"), ascorbic acid *plus* phenindamine tartrate, tripelennamine ("Pyribenzamine") hydrochloride or a placebo; among the 367 students studied satisfactory results were extremely rare. Some of the best results were claimed by the students themselves, from among those who had received a placebo only.

Rheumatoid Arthritis and Pregnenolene.

H. FREEMAN *et alii* (*The Journal of the American Medical Association*, April 15, 1950) describe the therapeutic effects of Δ^5-pregnenolene on rheumatoid arthritis in 11 men and 19 women between the ages of thirty-one and seventy-four years. Pregnenolene or pregnenolene acetate was given in doses of 300 to 700 milligrammes daily by mouth. Tablets of 100 milligrammes were used. Treatment continued for six to eleven weeks. Fifteen patients improved, but symptoms soon recurred when treatment was suspended. In general, improvement persisted for up to six weeks, which is longer than the period after ACTH therapy.

Rheumatic Fever and ACTH.

B. F. MASSELL *et alii* (*The New England Journal of Medicine*, April 27 and May 4, 1950) discuss the clinical response of rheumatic fever and acute carditis to ACTH. Ten patients with rheumatic fever were treated with 36 milligrammes for three days, 20 to 30 milligrammes for three weeks, 12 milligrammes for three days, six milligrammes for ten days and two milligrammes for ten days. Heart murmurs developed and disappeared during this treatment, including an aortic diastolic murmur. Mental depression occurred in some cases. In other cases symptoms decreased, but heart murmurs remained after treatment. In another group of cases 60 milligrammes of ACTH were given for three days, and 40 milligrammes for two weeks, with benefit in relation to joint pains, but auricular fibrillation persisted. The authors suggest that the effect of ACTH in rheumatic fever depends upon the stimulation of the adrenal cortex and a consequent increased production of 11, 17-oxysteroids, but the curative process is not understood. ACTH causes increased excretion of uric acid, decrease in the number of lymphocytes and eosinophile cells, and reduction in serum concentration of α globulin and in the titre of antistreptolysin O. In summary, the effect of ACTH was that the rheumatic process subsided within four to ten weeks in five cases, and in five it responded more slowly. Systolic and diastolic murmurs regressed or disappeared in three cases. congestive failure occurred in some cases, being at times relieved and at others aggravated by ACTH. Where ACTH caused an accumulation of fluids, control was easy with a diet of low sodium content and mercurial diuretics. Side effects were abdominal cramps, headache, swelling of the face, acne and skin striæ. The minimum effective dose of ACTH is between 20 and 50 milligrammes.

ACTH in Ophthalmic Conditions.

D. M. GORDON AND J. M. MACLEAN (*The Journal of the American Medical Association*, April 20, 1950) describes the use of adrenocorticotrophic hormone in certain eye diseases: corneal œdema, secondary glaucoma, chronic iridocyclitis, *retinitis pigmentosa* and acute chorioiditis. Some improvement was observed in four out of six patients, especially the two with iridocyclitis and chorioiditis. In general effects were temporary. The authors conclude that pituitary adrenocorticotrophic hormone may function to condition otherwise refractory patients to an excellent therapeutic response to some other subsequent treatment which is insufficient in itself to secure a result, for example, fever in uveitis. Glycosuria, hypertension and abdominal distension were noted temporarily in some patients during treatment.

JAMES OLSON *et alii* (*The Journal of the American Medical Association*, April 28, 1950) report results of the treatment of inflammatory eye diseases with ACTH. Seven patients with acute plastic iritis, keratitis, anterior uveal tract involvement, and secondary glaucoma were treated. All patients responded abruptly and favourably to doses of 185 to 432 milligrammes over a period of three to thirteen days. Glycosuria and auricular fibrillation were observed in two cases.

Rheumatic Fever and Ascorbic Acid.

B. F. MASSELL *et alii* (*The New England Journal of Medicine*, April 20, 1950) record their results in the treatment of rheumatic fever with ascorbic acid. Seven patients were treated with one gramme four times daily of ascorbic acid when suffering with rheumatic fever; they improved gradually in both symptoms and temperature and remained well. The sedimentation rate fell to normal in three cases. Generally there seemed to be a response to ascorbic acid by patients who did not respond to salicylates. However, the authors indicate that the cases studied are few, that the action of ascorbic acid, if any, is not understood, and that the relationship of ascorbic acid to adrenal cortex activity is unknown. They suggest further investigation.

BAL and Acute Lead Poisoning.

POUL BASTRUP-MADSEN (*The Lancet*, July 29, 1950) reports the treatment with BAL (dimercaprol) of two patients suffering from acute lead poisoning following ingestion of litharge. He states that in both cases the amount of lead excreted in the urine was considerably increased immediately after the institution of the therapy. The mobilization of lead did not seem to aggravate the symptoms of intoxication.

Digitalis Poisoning.

W. B. BURWELL AND J. P. HENDRIX (*The American Journal of Medicine*, May, 1950) state that the principal pharmacological effects of digitalis are an increase in the force of cardiac contraction, a slowing of conduction through the auriculo-ventricular node and an increase in myocardial irritability. The indications for and contraindications to the use of digitalis are based logically on the pharmacological properties of the drug; yet misuse occurs too frequently. The authors have collected 35 cases of poisoning by digitalis and related drugs from records and recent experience. Reports of eight cases are prescribed and commented on. The material collected is analysed from the standpoint of the reasons for digitalis therapy, the incidence of the signs and symptoms of poisoning with digitalis, and the causative factors leading to poisoning. The proper use of digitalis is discussed. The authors emphasize the need for sound knowledge, practice on the part of the physician, with the use of discrimination, judgement and caution, in the administration of digitalis, which they describe as a valuable but potentially dangerous agent.

Nitrogen Mustard Therapy in Lymphomata.

C. L. SPURR *et alii* (*The American Journal of Medicine*, June, 1950) review the clinical course in 95 cases of lymphoma and leuchæmia following treatment with methyl bis β-chloroethyl amine hydrochloride. These included 57 cases of Hodgkin's disease, 10 each of lymphosarcoma and chronic lymphatic leuchæmia, five of follicular lymphoblastoma, six of chronic myeloid leuchæmia and nine of other related diseases. The authors state that the clinical improvement and palliation in effects obtained, even when response was only brief, indicate the value of the therapy in Hodgkin's disease, lymphosarcoma and chronic lymphatic leuchæmia.

Toxicity of Carinamide.

W. P. BOGER AND J. W. CROSSON (*The American Journal of Medicine*, July, 1950) review the results of administration of carinamide to 1997 patients and state that the drug possesses a low order of toxicity. Exclusive of nausea and vomiting, the incidence of toxic manifestations was less than 2%. The nausea is considered to be due more to the mechanical factor of

swallowing large and frequently repeated doses of the drug than to toxicity. True drug sensitivity was confirmed in 14 cases and may be assumed in 23 more. There is no indication that carinamide interfered with the elimination of normal nitrogenous metabolites or permanently impaired renal function. It should be withheld in the presence of renal disease not because it damages the kidneys, but because it is unnecessary. It may inhibit the tubular excretion of some commonly used medications.

NEUROLOGY AND PSYCHIATRY.

A Short Method of Psychotherapy.

L. A. GOTTSCHALK (*The Journal of Nervous and Mental Disease,* October, 1949) has evolved a short method of psychotherapy. Many "difficult" cases are excluded from the list. The procedure is classified under the following headings: (i) ascertainment of the personality problem; (ii) bringing of awareness of personality mechanisms to the patient; (iii) modification of the behaviour patterns and psychological mechanisms; (iv) termination of the out-patient therapist relationships. The author is insistent on the need for creating a successful *rapport* with the patient. Treatment commences during the first interview. Patients must be seen an indefinite number of times with interviews of forty-five to sixty minutes.

Psychiatric Aspects of Vagotomy.

THOMAS S. SZASZ (*Archives of Neurology and Psychiatry,* December, 1949) discusses the reasons for the persistence of symptoms of gastric ulcer for which vagotomy has been performed, the ulcer having healed. He compares such ulcer pain to the pains of phantom limbs; in these conditions there is a persistence of the pain in the body image. He indicates the psychological explanation that may be responsible.

Arteriographic Study in Chronic Headache.

ARNOLD P. FRIEDMAN, EMANUEL FEIRING, LEO M. DAVIDOFF AND H. HOUSTON MERRITT (*Archives of Neurology and Psychiatry,* December, 1949) describe the effects of histamine and ergotamine tartrate injections on the calibre of the cerebral arteries rendered opaque by angiography. In two of the four cases in which histamine was injected, dilatation of the vessels could be demonstrated, and in two of the four cases in which ergotamine tartrate was injected, decrease in the size of the blood vessels was apparent. This report is intended to suggest a method of investigating the vascular basis of headache.

Clinical Variants of Moral Values.

GREGORY ZILBOORG (*The American Journal of Psychiatry,* April, 1950) draws attention to the modern tendency to approach every investigation with the attitude of objectivity and freedom from metaphysics, this being accepted then as unprejudiced, rational and accurate scientific study. He states that much in medicine is necessarily subjective, particularly those observations relating to human emotions—if the seeking of truth is the objective of science then such observation need be no less "scientific" for being subjective. Examples are given from the study of the sense of guilt, which, the author states, is not always due to a pathological state. Of recent years we seem to have fallen into the general error that all sense of guilt is neurotic, that all conscious reactions are super ego reactions, and that not to feel guilty is the ideal of normality. The purely clinical manifestations of moral values have as a rule nothing to do with real moral values; they are guilts expressed in terms of moral values. The sense of guilt of the depressed person who accuses himself of being worthless is perceived by him not for reasons of his having done something wrong, but for the reason of his having unconsciously wished to do something he unconsciously considered wrong.

General Principles of Psychotherapy.

LEO ALEXANDER (*The American Journal of Psychiatry,* April, 1950) states that much is conveyed by the manner and attitude of the doctor; perhaps less by what he says than by how he says it. He divides the types of technique of psychotherapy into seven main groups. (i) Supportive treatment, in which a complete and thorough history is taken and a complete and thorough examination is made, after which the doctor is convinced of the truth of his diagnosis and by word and deed can reassure his patient. (ii) Ventilation, which results in the patient's being interrupted as little as possible during the relating of his narrative. It is particularly useful in anxiety states of the higher age group. (iii) Abreaction, which is the release of tension associated with the history telling, sometimes achieved by direct urging, sometimes by the use of drugs such as ether, the barbiturates or "Benzedrine" sometimes by hypnosis. It is useful in cases of recent acute stress, particularly those in which the patient has reacted to such stress with profound inhibition. (iv) Shifting of the emphasis or the "flanking movement", in which the patient is not confronted with his conflict; but without much interpretation his attention is directed to a solution, and the emphasis is shifted from the past to the future. (v) Interpretation of an emotional illness for the patient, which requires the greatest amount of skill, tact and timing. The results are seldom dramatic, but rather a slow process of understanding resulting from continued cooperative efforts of patient and doctor. (vi) Suggestion, which is most successful in hysteria, and then only when attention is paid to the basic problem underlying the personality disturbance. It is far less powerful than is commonly assumed and should be positively expressed. (vii) Reassurance, which can commonly be helpful while an explanation of the patient's illness is presented to him, whether it be of emotional or physical origin. To be acceptable the doctor must have given sufficient time to convey that what he is saying is the truth. He must show an interest and concern for the patient's welfare, although it may not be possible to offer the hope of cure.

Drugs in Psychiatric Investigation.

BRYANT WEDGE AND MARGARET MOULTON (*The Journal of Nervous and Mental Disease,* February, 1950) state that they attempt to overcome the uncooperative attitude of many patients by the use of drugs which will produce "psychic analgesia", in which loquacity will take the place of taciturnity. A patient weighing one hundred pounds is given 1·32 grammes (two grains) of sodium amytal and 7·5 milligrammes of amphetamine sulphate forty minutes before the interview. The only undesirable side effects have consisted of uncomfortable drowsiness, dizziness or light-headedness in two cases. The authors claim that the method gives remarkable assistance in shortening the process of gathering information from verbally inhibited persons.

Group Therapy with Aphasics.

NATHAN BLACKMAN (*The Journal of Nervous and Mental Disease,* February, 1950) advocates the extended use of group psychotherapy in aphasic patients. He states that they are usually highly intellectualized persons, who suddenly find themselves in the position of a helpless child. Associated with the loss of speech there is usually a high degree of anxiety. The problem is not merely one of laborious speech reeducation, but also one of difficult psychotherapy. The author sees his patients in groups, encourages them to talk together, organizes group occupational meetings as in exhibition of handicrafts, and arranges activities to help one another in a practical way. In his summary of the more outstanding benefits he lists the following: (i) The aphasic loses his feeling of being isolated and of carrying a setback or disability greater than any other human has to carry through life. (ii) It gives the individual patient a feeling of successful, friendly competitiveness, of comparing both symptoms and aspirations in the presence of a benevolent parent substitute. (iii) It reaffirms his social acceptance, at least in terms of the group involved. (iv) It offers him an opportunity to give vent to accumulated hostility, to proceed to social concepts more tolerant to the given situation. (v) It strengthens the cognizance of himself, the aphasic, as a functioning, worthwhile, social human being. (vi) It lessens his need for dependency on parental figures and, as a corollary, for dependency on government benefits. (vii) It strengthens the individual in his task for persevering in the face of regressive tendencies. (viii) It provides the aphasic with a forum for testing progress, sharing experiences and enriching the ego-strengths so seriously battered in this category of disabled men.

Amnesia.

G. WILSON, C. RUPP AND W. W. WILSON (*The American Journal of Psychiatry,* January, 1950) state that amnesia for personal identification is a symptom which may occur with organic disease of the brain—namely, alcoholism, barbiturate poisoning, intracranial damage, psychoses (particularly schizophrenia) and hysteria, and frequently amnesia is deliberately simulated. The differentiation is clear in respect to ætiology in some cases. The presence of associated signs and symptoms and the preamnesic personality may be useful considerations in forming an opinion. In the opinion of the authors, if the amnesic episode seems to be a means of escape from a difficult personal situation or the consequences of this situation, then it is most likely simulated. In 59 cases almost half the patients were considered to be wilfully deceiving.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Saint George Hospital, Kogarah, New South Wales, on November 23, 1950. The meeting took the form of a series of clinical demonstrations by members of the honorary medical and surgical staffs of the hospital.

Pneumonectomy in Pulmonary Tuberculosis.

DR. M. STUART presented and discussed the X-ray films of a patient of DR. C. G. BAYLISS. The patient was a woman, aged twenty-five years, who in February, 1947, had been diagnosed as suffering from pulmonary tuberculosis affecting the left lung. On March 9, 1948, she was admitted to Waterfall Sanatorium, where pneumoperitoneum was attempted without benefit. On February 1, 1949, pneumonectomy was performed by Dr. J. McMahon. At the time of the meeting she was clinically well, and had been referred for rehabilitation training. An X-ray examination on October 5, 1950, had shown that the right lung was clear of abnormality. On the left side fibrothorax was developing.

Thoracotomy for Persistent Lung Collapse after Spontaneous Pneumothorax.

The second patient discussed by Dr. Stuart, also a patient of Dr. Bayliss, was a woman, aged twenty-six years, who on April 3, 1950, had had a spontaneous hæmo-pneumothorax on the right side. Blood was aspirated from the chest on two occasions. The result of a Mantoux test was negative. X-ray examination on April 20 showed a completely collapsed right lung with a little fluid present in the pleural cavity. It was presumed that there was still an opening on the surface of the lung which was unlikely to heal spontaneously. On May 12 bronchoscopic examination was carried out by Dr. Ian Monk. No abnormality was found. On May 16 thorascopic examination was carried out, but no definite leak was found. On May 22 thoracotomy was performed by Dr. Monk. A ruptured bleb was found and repaired. An X-ray film taken on July 12 showed partial expansion of the lung, and one taken on November 2 showed complete expansion.

Intussusception Associated with Carcinoma of Small Intestine.

DR. G. F. ELLIOTT presented a man, aged fifty years, who had been admitted to hospital under Dr. G. W. Ashby's care on August 30, 1950. The patient had been in his usual state of health until June, 1950; then he complained of pain occurring one hour after meals, mostly above the umbilicus. Some two weeks before his admission to hospital he began to vomit, mostly at night, with relief of the pain. The vomitus at times had a noticeably foul smell. He had become breathless and had lost weight. There was no change in the bowel habit. Examination of the patient revealed tenderness in the epigastrium, but no palpable mass. Examination of a fractional test meal showed very pronounced hyperchlorhydria, but no free acid and no macroscopic blood. Blood examination revealed a red blood cell count of 3,400,000 per cubic millimetre and a hæmoglobin value of 50%. The result of a benzidine test for blood was strongly positive. The gall-bladder appeared normal by Graham's test. The radiologist reported that there was a constant irregularity of outline high on the posterior wall of the stomach, with irregularity of the mucosal pattern apparently extending over an area of two to three square inches. He considered that the findings were very suggestive of a fairly early infiltrating carcinoma and that gastroscopy might be advisable. There was no definite evidence of ulceration, and the œsophagus and duodenum were normal in appearance. An opaque enema was seen to flow freely to the caecum, with no evidence of obstruction or filling defect. No diverticula were detected. On September 20 laparotomy was performed on a diagnosis of carcinoma of the stomach. The stomach was found to be normal. An intussusception of the jejunum was found, some six inches in length and about 12 inches from the duodeno-jejunal flexure. The intussusception was reduced easily, and a newgrowth was revealed. Some 24 inches of bowel were removed, and side-to-side anastomosis was performed. The pathologist made the following report on the material removed at operation.

> A piece of bowel approximately 24″ long with a puckering of its serosal surface in the mid length on the free border. The mucosal surface showed a raised tumour 2″ in diameter. One piece in section. Five enlarged lymph glands were located in the attached mesentery. Half of each in section. Microscopic: The section shows the tumour to be an adenocarcinoma infiltrating through the serosal surface of the bowel. The lymph nodes sectioned did not show any malignant involvement.

On October 11 the patient was discharged from hospital.

Large Gastric Ulcer in a Young Woman.

Dr. Elliott's second patient, a young woman, aged twenty years, had a history of mid-line epigastric pain of about one year's duration. The pain began about half an hour after meals and wakened her at night. It was relieved by ingestion of food and alkalis. She also complained of much flatulence, pronounced loss of weight and lassitude. During the two weeks preceding her admission to hospital, there had been great exacerbation of the pain and attacks of vomiting. She thought that she might have had melæna for one week. On admission to hospital she was very pale, miserable and apathetic, with obvious loss of weight. Her weight before operation was four stone ten pounds. A blood count on October 26, 1949, revealed a red cell count of 2,400,000 per cubic millimetre, a hæmoglobin value of 22% and a colour index of 0·45. On October 31, after transfusion of two litres of blood, the red cell count was 2,800,000 per cubic millimetre, the hæmoglobin value was 36% and the colour index was 0·6. X-ray examination showed a very large penetrating ulcer involving the lesser curvature of the stomach and the *incisura angularis*. There was no obstruction, but barium was held in the ulcer crater after the remainder of the stomach was emptied. At operation on December 14 the stomach was found to be large with an inflammatory mass, as large as a clenched fist, involving the lesser curvature, duodenum and pancreas. Adhesion was particularly dense to the pancreas. There were many enlarged glands. About three-quarters of the stomach and about eight centimetres of the duodenum were removed. Anterocolic anastomosis to the jejunum was carried out. Blood transfusion was given during operation, followed by saline and other intravenous therapy. Continuous Wangensteen suction was carried out for forty-eight hours. The pathologist submitted the following report on the material removed at operation.

> Stomach wall hypertrophied and shows an ulcer straddling the lesser curvature in its mid-length. The ulcer is 4 cm. in diameter and 2·5 cm. deep. One piece of ulcer in section. Several lymph glands varying in size from a small pea to a pigeon's egg were present in the attached piece of omentum.
>
> Microscopic: Sections show the ulcer to be a chronic peptic ulcer. The lymph glands show inflammatory hyperplasia. No evidence of malignancy was seen.

The patient was discharged from hospital on December 31 weighing five stone one pound.

Tantalum Gauze Repair of Incisional Hernia.

DR. T. E. WILSON presented a married woman, aged thirty-four years, who had seven children; she had had pyelonephritis with each pregnancy and toxæmia with the last three. In 1928 she had had scarlet fever. In 1933 she had undergone appendicectomy, which was followed by pyelonephritis, in 1948 hysterotomy, with termination of pregnancy and removal of the uterine tubes because of renal insufficiency (after that operation a slight sero-purulent discharge persisted from the wound for eighteen days), and in 1949 right ureterolithotomy. When admitted to the Saint George hospital on September 12, 1950, the patient stated that after the operation for sterilization in 1948 an incisional hernia had appeared and had gradually increased in size. She suffered from dyspnœa on exertion, but had had no œdema since 1948. On examination, she had a large reducible incisional hernia in the mid-line infra-umbilical wound with a wide divarication of the recti. Her blood pressure was 160 millimetres of mercury, systolic, and 110 millimetres, diastolic, and had been at or above that level for at least two years. On September 13 the incisional hernia was repaired with cotton sutures, and then a piece of tantalum gauze measuring six inches by two inches was fixed over the repair with braided tantalum sutures. The hernial sac was thick-walled and contained adherent omentum and small bowel. For two weeks after the operation there was some induration in the wound and the patient complained of tenderness over the upper edge of the gauze, but those effects subsided before she was

allowed out of bed on October 5. There had been no further symptoms referable to the wound or to the gauze, and the abdominal wall was quite firm.

Intussusception due to Trauma in an Adult.

Dr. Wilson's second patient, a young man, aged eighteen years, had been admitted to the Saint George Hospital on October 10, 1950. He stated that he had been in good health since a cholecystectomy seven months previously; but, at 5.45 p.m. on the day of admission to hospital he had been lying on the floor when a child jumped onto his abdomen. He immediately felt nauseated and complained of generalized abdominal pain. The nausea soon passed off, and he did not vomit. His bowels had previously been regular in action and had not acted between the time of the injury and his admission to hospital. When he was first examined at 10.30 p.m. the pain, which had steadily increased in intensity, was very severe and localized to the mid-abdomen. It did not radiate. On examination of the patient, there were guarding and tenderness on the right side of the abdomen, but no abdominal distension or palpable mass was present. The blood pressure was 140 millimetres of mercury, systolic, and 100 millimetres, diastolic, the pulse rate was 108 per minute, the temperature was 98° F., and the respiratory rate was 20 per minute. The provisional diagnosis was that of injury to the bowel, perhaps retro-peritoneal. At operation no increase was found in peritoneal fluid, nor evidence of bruising of the bowel or peritoneum. The lower part of the ileum contained an intussusception about two inches long, which was obviously of recent origin and which was easily reduced. After the operation he developed a mild paralytic ileus, which lasted for about four days. He was discharged to his home apparently quite well thirteen days after the operation. Since then he had had no further abdominal pain or other symptoms.

Ano-Rectal Fistula.

Dr. Wilson's third patient was a man, aged sixty-one years, who had been first operated on for an anal fistula in 1925. After that operation a slight perianal discharge persisted and a second operation was performed in 1946. The discharge ceased for a period of four weeks after the second operation, but it then recurred and had persisted despite a third operation in 1947. There was no history of tuberculosis or of chronic ulcerative colitis. On examination of the patient, the external opening of the fistula, which was surrounded by scar tissue, was found to be situated to the left of the mid-line about one and a half inches behind the anus. The right ischio-rectal fossa was indurated, and there was an area of induration under the mucosa of the posterior rectal wall at the level of the ano-rectal ring. No internal opening could then be found. At operation on May 31, 1950, the fistulous tracks including the internal opening into the bowel were laid open. This internal opening was found in the mid-line of the anal canal posteriorly between the pubo-rectalis muscle and the deep part of the external anal sphincter. From this opening a horse-shoe shaped submucous track extended forwards on either side of the ano-rectal ring. The main track extended backwards and then forwards again into each ischio-rectal fossa. The extension into the right ischio-rectal fossa was short, but that into the left ischio-rectal fossa expanded to form an abscess cavity and then passed forwards into the anterior prolongation of the fossa. After the fistulous tracks had been laid open, the edges of the wound were excised so that the wound was saucerized—it was then roughly circular and more than five inches in diameter. Gauze moistened with eusol was laid on the wound as a dressing (there was no cavity to pack). The dressing was changed after three days and then each day. After three months the wound was still two inches long and one-eighth of an inch wide. At the time of the meeting it was about one-quarter of an inch long and one-sixteenth of an inch wide. Dr. Wilson said that the final healing of the wound had been slow because of the fibrosis resulting from the healing of the large wound by second intention. However, for the first time in twenty-five years the patient was without his fistula, and there was no possibility of its recurring. Although the three parts of the external sphincter were divided, that had, of course, not resulted in incontinence, for the pubo-rectalis muscle was left intact.

Gastrectomy Plus Vagotomy.

The next patient presented by Dr. Wilson was a man, aged forty years, who had been admitted to the Saint George Hospital on September 12, 1950. He had suffered from a duodenal ulcer for twelve years. In 1938 he had had a hæmatemesis and had been treated intermittently since then with diet and antacids. For many years he had had attacks of vomiting, but those had been much worse during the current year. The patient did not know how much weight he had lost. X-ray examination after a barium meal on May 4, 1950, showed considerable narrowing and deformity of the pylorus with delay in emptying of the stomach, although no active ulcer was demonstrable. On examination of the patient on September 28, his general condition was good. There was a fixed tender spot on the epigastrium to the right of the mid-line, but there was no guarding of the muscles and no mass was palpable. A succussion splash was audible over the upper part of the abdomen. The hæmoglobin level was 12·6 grammes *per centum*, the red cell count was 5,000,000 per cubic millimetre, the blood urea content was 48 milligrammes per 100 millilitres, and the examination of a fractional test meal on October 5 showed hyperchlorhydria; the volume of the fasting specimen was 130 millilitres and that contained 50 millilitres of free acid. Because of the pyloric obstruction, the hyperchlorhydria, and the patient's age, partial gastrectomy *plus* vagotomy was performed on October 18, 1950. The procedure was "measured" gastrectomy (Visick), one inch of the lesser and two inches of the greater curvature being retained. The anastomosis was made with a long antecolic jejunal loop with the proximal end of the loop applied to the lesser curvature of the stomach. Two continuous layers of catgut and one interrupted layer of cotton sutures were used for the anastomosis. The lower end of the œsophagus was exposed, and an inch of both vagal nerves with their branches was excised. There were some peritoneal adhesions around the pylorus, which was almost completely stenosed, but no ulcer could be demonstrated. The stomach was very dilated. After the operation the remnant of the stomach was kept empty by aspiration for three days, and then the patient's diet was gradually increased. He was discharged to his home on November 4, receiving a Muelengracht diet and "Amphogel", and was free of symptoms. An insulin test meal examination on November 21 showed no response.

Intestinal Obstruction with Perforation.

Dr. Wilson next showed a married woman, aged thirty-seven years, who had had a left oophorectomy in 1938, a hysterectomy in 1945, a right oophorectomy in 1947, and an operation for the relief of a subacute intestinal obstruction due to adhesions in 1949. After the last operation she had been quite well till October 11, 1950, when she developed diarrhœa. On the following day she complained of severe colicky generalized abdominal pain and commenced to vomit. On October 3 the vomiting persisted, but the pain was less. During the next twenty-four hours her condition was satisfactory, but on October 4 there was a sudden deterioration. Her blood pressure fell to 60 millimetres of mercury, systolic, and 40 millimetres, diastolic, her skin was a dusky grey colour, she was sweating, and she complained of very severe generalized abdominal pain. Her abdomen was then rigid, and there was considerable tenderness, greatest in both iliac fossæ and in the pouch of Douglas. A plain X-ray film of the abdomen showed dilatation of the proximal coils of the jejunum. No fluid levels were seen. Gastric suction and blood transfusion were started; and an hour later her abdomen was opened through a lower transverse incision under local anæsthesia. Numerous peritoneal adhesions were present, and there was a diffuse peritonitis of the lower half of the peritoneal cavity. A gangrenous loop of ileum about two feet long, which contained an open perforation, was resected. The strangulation was due to a peritoneal band. After the operation the gastric aspiration and an intravenous infusion were continued for two days, and she was given penicillin (100,000 units three-hourly for nine days) and streptomycin (0·5 gramme six-hourly for nine days). After forty-eight hours administration of fluid by mouth was begun. Her bowels were not open for eight days. No enemata, aperients or parasympathetic stimulants were given after the operation. On the tenth day a fistula developed in the middle of the abdominal wound, and through this small bowel contents discharged. With the use of posture, increased salt intake and a buffer jelly, gross excoriation of the skin was avoided, and after four week the fistula closed spontaneously. At the time of the meeting, her bowels were acting normally, and her general condition was rapidly improving.

Local Amputation for Diabetic Gangrene.

Dr. Wilson's last patient, a man, aged seventy-two years, had been admitted to the Saint George Hospital on May 30, 1950, because of *diabetes mellitus* and gangrene of two toes. Six weeks previously he had had an infected left hand, which was slow to heal. For one month he had complained

of increasing pain of a burning quality in his left foot. During that time he had noticed black areas on the adjacent parts of the left third and fourth toes, and those areas had gradually extended. When he was admitted to hospital the adjacent parts and the dorsa of the left third and fourth toes were gangrenous, and the distal part of the dorsum of the left foot and the medial four toes were acutely inflamed. There was a small amount of thin purulent exudate from the edges of the gangrenous areas. Both popliteal arteries and the right posterior tibial artery were palpable and pulsating, but no pulsation could be detected in the vessels of the left foot. His general condition was good for his age, the blood pressure was 170 millimetres of mercury, systolic, and 120 millimetres, diastolic, the pulse rate 80 per minute, and the temperature 98° F. The affected toes were dressed with penicillin powder and covered with a sterile dressing. Administration of penicillin, 100,000 units six-hourly, and of streptomycin, 0·5 gramme eight-hourly, was started. The penicillin was continued for six weeks, and the streptomycin was given for one week at first and then a second week later after the amputations. An 1800 Calorie diet was given at first and later increased to 2000 Calories. Protamine zinc insulin was gradually reduced from 25 units per day to 10. His diabetes was well controlled. On the day after his admission to hospital alcohol injections of the left lumbar sympathetic chain were given, and after this the lower part of the left leg and foot were warmer than the right. The inflammation of the left foot and toes subsided after two weeks, and there was no further extension of the gangrene. On June 28 amputation of the left third and fourth toes was carried out with the patient under "Pentothal" anæsthesia. The operation was followed by some sloughing of the skin edges, and on July 26 those were excised.

Dr. Wilson said that at no time since then had the patient been pyrexial, and there had been no recurrence of the inflammation in his left foot. The amputation wound had been slow to heal, but it was now dry. The circulation in the other toes of the left foot was very poor. He had been able to walk without pain since the beginning of September, when he was permitted to bear weight on his left foot.

Bilateral Hydroureter.

DR. R. INNES CAMPBELL presented a man, aged twenty-five years, who had first attended the out-patient department at the Saint George Hospital on February 7, 1950, being referred by Dr. A. M. Macintosh, of Cronulla. His only complaint was of a dull aching pain in both sides of the back in the lumbar region, but worse on the right side, for about three months. He had had occasional severe attacks of pain on the right side. The pain did not radiate to either groin. He had no urinary or any other symptoms. There was slight tenderness in both loins, but no palpable enlargement of either kidney. Cystoscopic examination showed pin-point ureteric orifices on both sides, with ballooning up of the vesical mucosa around the orifices at each efflux—in other words, bilateral ureteroceles were present. An excretion pyelogram showed bilateral hydronephrosis and hydroureter, the ureters being tremendously dilated right down to the bladder, where they suddenly narrowed to almost nothing. There was immediate function of both kidneys, but very little dye had entered the bladder after twenty-five minutes. It was decided to try the effect of hypodermic injections of pituitrin, one millilitre daily for six weeks, but that had no effect at all. His symptoms still persisted, and an excretion pyelogram on March 14 showed the hydronephrosis and the hydroureters to be slightly larger. He was readmitted to hospital, and on April 15 both ureteric orifices were slit about half an inch by means of cystoscopic scissors. Both ureters were then catheterized, and the urine from each was examined and cultured. *Proteus vulgaris* was grown on culture. Before the ureters were split, it was impossible to pass a catheter through the orifices. On May 2 a further cystoscopic examination was carried out, and no stenosing of the ureteric slits had taken place. He was not seen again until November 7, when he had a further excretion pyelogram; he said he had not reported back to the hospital as he had felt so well, and was doing very heavy work. On that date the ureters were almost back to their normal size, but there was still a little hydronephrosis of each kidney and slight clubbing of the calyces. There was, however, quite a fair amount of dye in the bladder within ten minutes. Dr. Campbell said that the man's condition was probably a congenital one, but it was difficult to understand why he had had no symptoms until he was twenty-five years of age.

Total Cystectomy for Carcinoma of the Bladder.

Dr. Campbell then presented a man, aged thirty-six years, a poultry farmer, who had first attended the out-patient department at the Saint George Hospital on June 15, 1948. He complained that he had first passed blood in his urine eight years previously. There had been only a little, and the symptom had passed off quickly. This had happened on a "number of occasions". When he passed the blood he had a burning pain in the penis. He also had frequency of micturition during the attacks of passing blood. He had no other symptoms, and on examination of the patient nothing abnormal was found in the alimentary, cardiac, respiratory or central nervous system. On June 19 cystoscopic examination revealed a very large tumour on the right side of the bladder about the region of the right ureteric orifice. Its appearance was that of a large papilloma. On account of its size, it was decided that it could be dealt with only by open operation. On June 23 a suprapubic cystostomy was performed, and a very large pedunculated papilloma was removed by diathermy excision. The patient's convalescence was uneventful, and he was discharged from hospital on July 17 with clear urine and no urinary symptoms. The pathologist, Dr. A. E. Gatenby, made the following report on the tumour:

> A soft cauliflower-like tumour, 2″ by 1½″ by 1″, with a pedicle ¾″ in length. One piece of tumour and adjacent pedicle were sectioned. Sections show the tumour to consist of papillary processes of transitional epithelium, with thin fibrovascular cores. The appearances are those of a villous papilloma. Occasional mitotic figures are present, and there is some invasion of the pedicle by epithelial downgrowths. However, the papillary architecture is well preserved, the cells for the most part are orderly, and I do not think there is any definite evidence of malignancy.

Follow-up cystoscopic examinations were carried out on September 24, 1948, and January 15, 1949, when, apart from some scarring about the right ureteric orifice and the right side of the trigone, the bladder appeared quite normal. The patient had been free of all urinary symptoms. He did not have a further cystoscopic examination until November 2, 1949, when he reported that he had been quite well, and had not had hæmaturia, scalding or frequency. However, examination showed multiple recurrences about the original site and one near the apex of the bladder. They were flat and sessile and not pedunculated. The condition now was very suggestive of carcinoma, but it was decided to attempt further treatment by cystoscopic fulguration. He was also given weekly 10 millilitre injections of podophyllin for the next six weeks. That treatment had no effect on the tumours. Further fulguration of the tumours was carried out on December 17, 1949, and on January 21, March 11, April 29 and July 21, 1950, but the results were not encouraging.

From December, 1949, his original symptoms returned, and were gradually growing much worse. He had hæmaturia off and on and frequency of micturition sometimes half-hourly. He also complained of "pain in the bladder" and along the penis on micturition and of passing small pieces of "fleshy material". Despite extensive fulguration of the tumours on each occasion, their condition was not improved. They were showing no response whatever to fulguration. In view of the severity of his symptoms, and the lack of response to treatment, it was decided that much more drastic methods must be adopted. The multiple tumours and their position precluded partial cystectomy, and it was decided that total cystectomy was the only operation worth attempting. The patient readily agreed to have that performed. Before operation an excretion pyelogram was performed, and it showed, as was to be expected, a moderate degree of dilatation of the right ureter and right renal pelvis. There was no dilatation of the left ureter or left renal pelvis. Function of both kidneys was quite good. The operation was carried out in two stages, the right ureter being transplanted first into the pelvic colon on October 7, 1950, and the left one similarly transplanted and the bladder removed on October 28, 1950. The pelvis was carefully examined for glands, but none were seen or felt. The transplants were carried out by the Coffey method, with the modification of Bernard Ward. For twenty-four hours after the first operation the patient was given a continuous intravenous drip administration of normal saline and glucose solution, alternating with 4·2% sodium sulphate solution. His convalescence was very smooth, and he was quite fit for the second operation three weeks after the first. During the second operation he was given a drip blood transfusion, and after operation he was again given intravenous therapy with saline, glucose and sodium sulphate solutions for twenty-four hours. Both operations

were carried out under a cover of penicillin and sulphadiazine, both those drugs being given a week before and a week after each. The patient's convalescence after the second operation was just as smooth as after the first. For a few days he had very frequent bowel actions of urine and fæces, but ten days after operation he had a three-hour control. He was allowed out of bed on the fifteenth day, when his wound was healed, except for a slight discharge from the lower end of the wound. The time between his bowel actions had then increased to about four hours.

Dr. Campbell said that the improvement in the patient's condition was most remarkable. He looked a new man, and was extremely grateful to be relieved of his continuous pain and burning. For that alone the operation was completely justified, whatever the ultimate result might be. Dr. Gatenby, the pathologist, had again examined the tumours, and reported that their main structure was that of villous papilloma, but with early adenocarcinomatous changes; there was no deep penetration of the pedicle of one tumour examined.

A further excretion pyelogram was to be carried out in two or three months' time, and it was proposed to arrange a course of deep X-ray therapy.

Bilateral Hydronephrosis.

In presenting his last patient, a married woman, aged thirty-three years, Dr. Campbell said that her case presented an interesting problem from two points of view: (i) the distinction between an enlarged gall-bladder and hydronephrosis, and (ii) the problem of treatment in gross bilateral hydronephrosis. The distinction between an enlarged gall-bladder and a movable kidney or hydronephrosis might seem to offer no difficulty. The diagnosis might have to be made purely by palpation. Often there was no jaundice to suggest gall-bladder trouble, nor need there be any obvious urinary change to suggest kidney lesion.

The patient had first complained of a dull aching pain in the right loin at the age of thirteen years. Her appendix was removed at the age of fourteen years. At the age of sixteen years she complained of pain, mainly in the right hypochondrium. She had attacks of colic at intervals of six to twelve months on and off. She was given a surgical corset with no relief. Her attacks became more frequent over the following years. Two years before the meeting she was examined when she had a swelling the size of a swan's egg in the right hypochondrium. It was apparently difficult to decide whether the swelling was gall-bladder or kidney. A Graham's test revealed a non-functioning gall-bladder. Her gall-bladder was removed in February, 1948. Multiple calculi were present. Since then she had had further attacks of vomiting and colic associated with a similar swelling visibly distending the abdominal wall. It was only the attack on February 28, 1950, that revealed any evidence of kidney involvement. Increased pain was accompanied by increase in the size of the tumour and diminution of pain followed by polyuria. Palpation also gave a peculiar sickening sensation.

That was the position, Dr. Campbell said, when the patient came to the urological ward in February, 1950. The pain was very severe, and the attacks would last from two to four days; morphine one-quarter of a grain was required up to three times daily. Excretion pyelogram and right and left retrograde pyelograms were prepared at that stage. In plain X-ray films both renal outlines were enlarged; no stones were seen. In urograms good shadows appeared on the left side in seven minutes; renal function on the right side was delayed. A twenty-five-minute film showed poor bilateral renal excretion as indicated by the small amount of drug in the bladder. A huge hydronephrosis was demonstrated on the right side. On the left side a large hydronephrosis was present, but not of the same degree of severity as on the right. No drug was seen in either ureter. Both ureters were catheterized and retrograde pyelograms were prepared. The latter confirmed the findings of the excretion pyelogram. Examination of the urine showed granular casts in the urine from the right kidney, and from the left there were pus cells and a few red cells. *Staphylococcus aureus* was grown on culture from the urine from the left kidney. After intravenous injection of indigo carmine no blue colour appeared from the right kidney after eleven minutes. The catheter passed to the left kidney without obstruction, and a good blue colour was obtained from that side after four minutes, the left renal pelvis having been emptied with a syringe. The patient's blood urea content was 48 milligrammes per 100 millilitres. On March 23, 1950, the left kidney was explored. There was a well-marked constriction half an inch below the pelvi-ureteric junction. A plastic operation was performed. The upper end of the ureter was slit longitudinally into the renal pelvis and then sutured transversely. A pyelostomy tube was inserted into the renal pelvis, and a ureteric catheter was passed through the renal pelvis into the upper part of the ureter. Both were drained through the loin for two weeks. Convalescence was very smooth and satisfactory. On June 1 a further excretion pyelogram was prepared, and it was found that compared with the X-ray appearance on the previous occasion there was a definite improvement in the condition of the left kidney. There was still considerable dilatation of the renal pelvis, but there was not nearly so much dilatation of the major calyces, which were beginning to show a cupped appearance. The condition of the right kidney appeared to be much the same as before. The patient continued to have further attacks of right-sided pain, and she was readmitted to hospital on October 13. Cystoscopic examination was carried out, with bilateral ureteral catheterization and intravenous injection of indigo carmine. The catheter was passed to the left kidney and a good blue colour appeared in six minutes. Examination of a wet film of urine from the right side revealed a moderate number of pus cells, and occasional red cells. No abnormal constituents were seen in a wet film of urine from the left side. On culture of urine from each side, no organisms were recovered. It was decided then to explore the right kidney. At operation a greatly dilated renal pelvis was found to be present. The ureter was adherent and curled around the pelvis, and only a shell of renal tissue was left. A right nephrectomy was carried out. Convalescence was uneventful, and the patient was very well at the time of the meeting.

(To be continued.)

THE OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA.

THE tenth annual general meeting of the Ophthalmological Society of Australia (British Medical Association) was held in Brisbane in conjunction with the meetings of the Section of Ophthalmology of the Australasian Medical Congress (British Medical Association) on May 29, 1950.

The following office bearers were elected for the ensuing year: *President*, J. Bruce Hamilton; *Vice-President*, G. A. Brew; *Honorary Treasurer*, A. E. F. Chaffer; *Honorary Secretary*, A. L. Lance.

It was decided that the eleventh meeting of the society be held at Hobart on November 12, 1951.

Special Correspondence.

NEW ZEALAND LETTER.

FROM OUR SPECIAL CORRESPONDENT.

Two Acts of medical interest have been passed in the 1950 session.

Medical Research Council Act, 1950.

The *Medical Research Council Act*, 1950, incorporates the Medical Research Council, which has been in existence since 1937, in order to assure continuity of medical research, and permanent conditions for research workers. It is hoped also to attract endowments and bequests to assist in medical research. The membership is to include the Director-General of Health, the permanent secretary of the Department of Scientific and Industrial Research, the Dean of the medical faculty at the University of Otago, a series of nominees to be appointed by the Minister from the British Medical Association, the Board of Health, the New Zealand Committee of The Royal Australasian College of Physicians and the Royal Australasian College of Surgeons and the Royal Society of New Zealand, and two from the Senate of the University of New Zealand recommended by its academic board. The functions of the Council are to foster medical research, publish reports and furnish advice. It may make grants to individuals, colleges and institutions from a fund to be vested in the Council, the Medical Research Endowment Fund. Any discoveries *et cetera* made by officers of the Council or by those working under its support are to be the property of the Council.

These provisions mark a major step forward, and follow soon after the visit of Sir Henry Dale and others whose advice the Government sought. During the discussion of the bill in Parliament strong representations were made on the importance of dental research, and the inclusion of a dental representative on the Council. The Minister (the Honourable J. T. Watts) announced that it was planned to appropriate £33,000 from government funds this year, as against £21,000 last year. This was in contrast to the total sum of £25,000,000 to £30,000,000 spent last year, *in toto*, on the health of the community.

Medical Practitioners Act, 1950.

The *Medical Practitioners Act*, 1950, consolidates previous Acts relating to registration and discipline, and adds a number of new conditions, chiefly those recommended by the Cleary Report of 1948 touching discipline under the *Social Security Act*. (See THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1949, page 730.)

The Medical Council remains much as before in constitution and powers, and is constituted a higher court in relation to the Disciplinary Committee. It consists of seven members—the Director-General of Health, the Dean of the Medical Faculty, and five practitioners appointed by the Minister, one on the recommendation of the British Medical Association. Its powers are to impose a fine up to £100, to bring about suspension from practice for up to twelve months with the consent of the Attorney-General, and to initiate a motion to the Supreme Court to remove the practitioner's name from the register. A new variety of registration is introduced (to be effective at a date to be gazetted), namely, conditional registration for recent graduates, to be confirmed as registration after a period of internship or other service similar to that required in England. Reciprocity with other countries remains as before.

Within the profession, discipline is vested in a Medical Practitioners Disciplinary Committee and in Divisional Disciplinary Committees. All the members of these committees are medical men, and in almost every case are to be appointed by the British Medical Association. Their scope is professional misconduct, short of grave impropriety or infamous conduct in a professional respect, which is the concern of the Medical Council. The powers of the Disciplinary Committee are subject to appeal to the Medical Council, and include the imposition of a fine up to £100, censure, or the giving of an order to pay costs and expenses.

Nutrition and Dietetics.

Also passed in the 1950 session was an Act to further the knowledge of nutrition and dietetics and to provide for the training and registration of dietitians under the Dietitians Board.

Qualification for registration is defined by regulations, and power is given to the Board to approve hospitals and other institutions as training schools. Registered dietitians are to have annual practising certificates, and hospital boards may appoint only registered dietitians.

The Board has disciplinary powers over registered dietitians subject to appeal to a special magisterial Board of Appeal. The uniform and the title "dietitian" are protected under the Act.

Correspondence.

TREATMENT OF EXTERNAL OTITIS.

SIR: I am writing to make appraisal of a comparatively new treatment of external otitis, which, as every practitioner knows, can prove very resistant to the recognized therapeutic methods. I refer to the local streptomycin applications, for which statistics were compiled by Senturia and Broh-Kahn in the *Annals of Otology, Rhinology and Laryngology*, in March, 1947. The latter workers were able to isolate Pseudomonas organisms from almost 100% of infected ears, but found it to be absent from the normal external meatus. Upon this change in the meatal flora the treatment is based.

Unfortunately we have not been able to identify the organisms with the same frequency, but the clinical results of treatment have been very gratifying. In a series of 42 cases treated by this method, all have shown marked improvement after three or four applications. In 27 of these the discharge cleared up entirely, and any residual itch or soreness had cleared up within a week of cessation of treatment. In the remaining 15, some minor manifestations persisted for varying periods, such as a slight itch or an isolated tender spot on the meatal wall, but in no case did the discharge persist. In the latter group a fungus could be isolated in three cases, and in four others there was evidence of eczema elsewhere.

Following are the details of the method. Streptomycin ointment 0·5% is warmed slightly in a small crucible and is used to impregnate a half-inch gauze wick, which is then inserted into the meatus so as to thoroughly contact the inflamed area. The efficient introduction of the wick is of the utmost importance, and the great majority of failures are due to inadequate practice or lack of diligence in this matter. The wick is changed daily or every other day, according to the severity of the condition. In very acute conditions associated with adenitis, a few days' intramuscular antibiotic therapy should be given, or, in the case of ample supplies becoming available, aureomycin, 250 milligrammes four times daily for three days, is sufficient.

It is hoped that this assessment might be of some value to the general practitioner, who encounters this troublesome condition so often in the warm moist climate prevalent throughout our coastal area.

Yours, etc.,

215 Macquarie Street, Sydney,
Undated.

HARVEY LISTER.

THE EMPIRE MEDICAL ADVISORY BUREAU.

SIR: On my return from London I would very much like to express my appreciation of the help I received from the British Empire Medical Advisory Bureau.

Both Dr. Sandiford, the director, and Miss Jarvis, his secretary, were more than helpful. It was through them I was able to secure a most comfortable flat in London.

Mrs. Langley and myself thoroughly enjoyed the "teas" which we attended at Tavistock Square and meeting our colleagues and their wives from various parts of the Empire.

Yours, etc.,

Delegate, New South Wales,
November 24, 1950.

ALAN L. LANGLEY.

THE MENSTRUAL PERSISTENCE OF ALLERGIC SYMPTOMS AND THE INFLUENCE OF PREGNANCY ON THEM.

SIR: The text-books commonly refer to two behaviour characteristics of allergic symptoms as being generally recognized. Firstly, these symptoms tend to be aggravated during the premenstrual or menstrual period, and secondly, it is usually said that they are relieved during pregnancy, although Cooke suggests that aggravation and relief appear with equal frequency in his cases.

One not infrequently encounters patients whose symptoms are relieved by routine methods of treatment, except that they continue to be troubled with their premenstrual attacks. In 1933 Rasmussen showed that the enlargement of the pituitary, which was known to accompany pregnancy, was confined to the anterior lobe, and this work came under my notice at about the time that I had under my care some patients suffering from such premenstrual or menstrual persistence of symptoms. It was also known at that time that the anterior lobe of the pituitary exerted a potent influence on the cyclic activity of the ovary, so trial was made with treatment by the only preparation then available, which was an extract of the whole anterior pituitary substance. Injections of one millilitre were given on the ninth, sixth and third days before the next expected menstrual period, and these first patients showed considerable immediate improvement, which was maintained by repetition of the course for several premenstrual periods.

When the newer preparations made from pregnancy urine became available, they were used with almost uniform success. At first it was thought that they might not prove to be so potent as the older preparation, so a course of four doses was used, one millilitre being given on the tenth, seventh, fifth and third days before the expected menstrual

period. This was later reduced to three doses without any loss of efficiency, and the course is repeated for three or four menstrual periods.

In going through some clinical histories recently, I found 57 patients who complained of menstrual aggravation of symptoms, and of these 18 had premenstrual persistence after considerable or complete relief of their intermenstrual attacks had been achieved. Of this series of 18 patients, 17 were relieved by an anterior pituitary preparation and one was not relieved. Those relieved included three suffering from eczema, two from migraine, one from intermittent hydrarthrosis, and the remainder from respiratory allergies.

It must be emphasized that this form of treatment has been found to be of use only in those cases in which the intermenstrual manifestations have already been alleviated by standard methods of treatment, and the main complaint is now the recurrent attack at or just before the menses. It has been tried as a general form of treatment, both with and without ascorbic acid, but without success.

In the history taking I had personally gained the clinical impression that pregnancy was accompanied by improvement in the majority of cases, and that therefore my experience was in conformity with the view recorded in the majority of text-books. However, in going through this series, I found that of 49 patients who gave information concerning the influence of pregnancy, 28 stated that their condition was worse, and only 18 that improvement had occurred; the remaining three said that their condition had been improved during some pregnancies and worse during others. I feel that my false clinical impression on this point is possibly due to the fact that the patient whose condition has been improved tells about it early in her story and stresses it, whereas the patient whose condition has been worse during her pregnancies tends to accept that state of affairs as a natural consequence, and does not spontaneously volunteer any information on the point.

Yours, etc.,

Perth, Western Australia, November 20, 1950.

H. G. D. BREIDAHL.

Bibliography.

Bray, G. (1937), "Recent Advances in Allergy", Third Edition, page 46.

Coke, F. (1923), "Asthma", pages 114, 124.

Cooke, R. A. (1947), "Allergy in Theory in Practice", page 551.

Rasmussen, A. T. (1933), "Percentage of Different Types of Cells in the Anterior Lobe of the Hypophysis in the Adult Human Female", *The American Journal of Pathology*, Volume IX, page 459.

Urbach, E., and Gottlieb, P. M. (1946), "Allergy", Second Edition, page 57.

Obituary.

PAUL TILLETT.

WE are indebted to Dr. Douglas Carruthers for the following appreciation of the late Dr. Paul Tillett.

Paul Tillett passed away peacefully on October 31, 1950, while at sea. To his many friends changes of grim portent had been apparent for some time. The shock, nevertheless, was great, for he was but fifty-four years of age. Paul knew that he was threatened, yet his courage never wavered, and he remained enthusiastic, enterprising and always cheerful. He had set his mind to a long restful voyage overseas.

A professional man, like the head of a business, cannot suddenly drop the reins, and for Paul, with a big radiological practice, it meant careful planning, and it took time. At last the day did come, and, cheerful with the weight of responsibility lifted from his shoulders, he set sail from Sydney on October 17. Alas, the grim reaper spared him for only two weeks, for he died before the ship reached Cape Town, its first port.

Paul will be widely mourned, for he had many friends in the professional, business and sporting world. He was one of those with whom his friends always welcomed the

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED NOVEMBER 25, 1950.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	..	1	..	..	..	..	..	1
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	..	1(1)	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	..	..	..	..	..	..	..
Diphtheria	6(6)	10(7)	3(3)	..	..	..	..	..	19
Dysentery (Amœbic)	*	..	..	1	..	..	..	..	1
Dysentery (Bacillary)	*	1(1)	..	..	..	..	*	..	1
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	1	*	*	..	..	1
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	1	*	*	..	*	*	..	1
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	..	..	..	..	..	..	..	..
Measles	*	*	*	376(193)	*	*	..	..	376
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	29(9)	2	10	13(13)	1(1)	8(2)	..	1	64
Psittacosis	*	*	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	..	..	1	..	..	1
Rubella(*c*)	*	*	..	..	1(1)	*	..	1	2
Scarlet Fever	46(18)	28(20)	36(32)	9(5)	5(4)	3(3)	..	..	127
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	1(1)	..	*	*	*	*	*	1
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	26(17)	22(14)	16(5)	11(9)	12(9)	7(2)	..	..	94
Typhoid Fever(*e*)	..	..	1(1)	1(1)	..	1(1)	..	..	3
Typhus (Endemic)(*f*)	..	..	2	..	1(1)	..	..	..	3
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	*	*	1	*	*	*	*	*	1
Whooping Cough	*	*	*	11(10)	*	*	..	..	11
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from the Northern Territory.

* Not notifiable.

(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

opportunity to stop and talk. He moved in a wide circle and was always well informed. His conversation was fluent, often spiced with a delightful wit, or when the need arose, offering words of sound judgement or serious kindly advice.

Paul Tillett was educated at Sydney Grammar School and at the University of Sydney, where he graduated in 1920. After study overseas he practised in radiology, first at Orange, New South Wales, and then in Sydney. He was honorary radiologist at the Royal North Shore Hospital of Sydney, the Prince Henry Hospital and the Balmain and District Hospital. In his practice he was untiring and ever obliging to patients and to his colleagues. He combined skill in radiological technique and interpretation with a profound clinical knowledge, so that every case was of interest to him. His diagnostic opinion was always worth listening to.

Paul served as major in the Australian Army Medical Corps in the recent war. In the world of sport, golf and deep-water fishing were his major loves. He was a member of the Australian Golf Club and in his earlier years played from a single figure handicap. For some years he sailed his own yacht and was often to be met at his favourite resort, Jervis Bay, where he knew every rock and reef, and to those who went with him he seemed to know, too, just where the fish were schooling.

Friends, colleagues and acquaintances will deeply mourn his passing. All extend heartfelt sympathy to his wife, Hope, and to his little daughter, Paula.

EDWARD FIELD.

WE regret to announce the death of Dr. Edward Field, which occurred on November 28, 1950, at Glenhuntly, Victoria.

JOHN IRVING GUENTHER.

WE regret to announce the death of Dr. John Irving Guenther, which occurred on November 28, 1950, at Adelaide.

Notice.

EXAMINATIONS FOR THE FELLOWSHIP OF THE FACULTY OF RADIOLOGISTS, LONDON.

EXAMINATIONS for the Fellowship of the Faculty of Radiologists, London, will be held in Australia in May, 1951. The examination will be conducted by the Faculty of Radiologists, in association with the College of Radiologists (Aust. and N.Z.), and will consist of examinations in medicine, surgery, pathology and either radiodiagnosis or radiotherapy. The Faculty of Radiologists has appointed Professor B. W. Windeyer, of the Middlesex Hospital, and Dr. M. H. Jupe, of the London Hospital, to visit this country as examiners for this purpose. Further details and application forms for intending candidates can be obtained from the Honorary Secretary, the College of Radiologists (Aust. and N.Z.), 135 Macquarie Street, Sydney.

Bibliography of Scientific and Industrial Reports.

THE following bibliographies, summaries of information and special reports have been prepared by the Commonwealth Scientific and Industrial Research Organization Information Service. Copies may be obtained on application to the Officer-in-Charge, C.S.I.R.O. Information Service, 314 Albert Street, East Melbourne, C.2. The bibliographies are, in the majority of cases, selective only. Applicants should state clearly the reason the bibliography *et cetera* is requested, because the number of copies available is limited.

B.425: "Benzedrine—Qualitative and Quantitative Analysis—Selected References", August, 1950 (seven references).

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Streimer, Ludwig, registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1945, 16 Botany Street, Bondi Junction.

Angel, Henry Reece Tennant, M.B., B.S., 1950 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Diary for the Month.

JAN. 3.—Western Australian Branch, B.M.A.: Council Meeting.
JAN. 9.—New South Wales Branch, B.M.A.: Council Quarterly.
JAN. 11.—South Australian Branch, B.M.A.: Council Meeting.
JAN. 12.—Queensland Branch, B.M.A.: Council Meeting.
JAN. 16.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JAN. 18.—Victorian Branch, B.M.A.: Executive Meeting.
JAN. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JAN. 24.—Victorian Branch, B.M.A.: Council Meeting.
JAN. 26.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney)—All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

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